THE

WEST RIDING LUNATIC ASYLUM

MEDICAL REPORTS.

VOL. III.

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'For does not health mean harmony, the synonym of all that is true, justly-ordered, good ; is it not, in some sense, the net-total, as shown by experiment, of whatever worth is in us? The healthy man is the most meritorious product of Nature, so far as he goes. A healthy body is good; but a soul in right health,—it is the thing beyond all others to be prayed for ; the blessedest thing this earth receives of heaven.'   CARLYLE.

PREFACE.

It is hoped that the Third Annual Volume of 'West Riding Lunatic Asylum Medical Reports' will be received as a proof that the Medical Officers of that large hospital are earnestly endeavouring to avail themselves of the vast opportunities which it affords to give breadth and precision to our knowledge of mental diseases, and to multiply our means of alleviating them and the miseries which they entail. There can be no presumption in believing that the volume contains some important contributions to medical literature. Nor can there be any unworthy pride in feeling gratified that several of these are from the pens of distinguished men, who, although not officially connected with the West Riding Asylum, have yet associated themselves with it by their labours, and have thus recognised its claim to be regarded as a centre of vigorous effort in the interests of science and humanity.

The firm conviction which is entertained that a sound system of mental hygiene, and improved methods of treating mental aberrations, are among the most pressing wants of the day, and that these may be reached by patient study and research, has animated and directed the work which is

here recorded, and will continue to stimulate to further diligence and exploration.

Although considerably larger than those which have preceded it, this volume is still quite inadequate to contain the materials which have sought a place in it. Five very valuable essays, containing original observations made in the wards of the asylum, have, therefore, been regretfully, but unavoidably, excluded from it.

CONTENTS.

CONVOLUTIONS OF THE HUMAN BRAIN

IN RELATION TO THE INTELLIGENCE.[1]

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THAT the Brain is the organ of the Mind; that the convolutions forming the surface of the hemispheres of the cerebrum are the parts where the mechanism or apparatus is situated which furnishes the material conditions through which the intellectual processes are manifested; and that every mental act is associated with some molecular change in the grey matter of the convolutions, are propositions universally admitted by physiologists.

In this Essay I propose to give a general account of the method of construction of these convolutions, and to point out the mode in which their constituent parts are put together and arranged.

My remarks will be grouped under the following heads :—

> MASS AND WEIGHT.
> EXTERNAL CONFIGURATION.
> INTERNAL STRUCTURE.
> VASCULAR SUPPLY.

[1] This Essay was delivered as a lecture at a Medical Conversaziono in the West Riding Asylum, October 15, 1872. In preparing it for the press, I have added various numerical and other details, which for want of time could not conveniently be introduced into an oral discourse.

Mass and Weight.—One almost insensibly associates the ideas of power and strength with size and weight, and when one sees a large-headed, big-brained man, one is disposed to say that such a person must be endowed with great intellectual capacity. This very general and popular conception of the relation between size of brain and degree or force of intellect to some extent undoubtedly rests on a basis of truth. It is supported by the well-known examples of Cuvier, Dr. Abercrombie, Daniel Webster, and Dr. Chalmers, in whom the brain-weights were ascertained to be, respectively, 64·5 ozs., 63 ozs., 53·5 ozs., and 53 ozs.; and by the more recent, and therefore less widely known, examples of Professor Goodsir and Sir J. Y. Simpson, whose brains weighed, respectively, 57·5 ozs. and 54 ozs.—weights which even in the least heavy of these brains are considerably above the general European male average of 49 or 50 ozs.

But that the size and weight of the brain are accurate measures of the degree of the intelligence is a proposition which must not be accepted too absolutely, and in many cases at least requires considerable qualification. To take an illustration from comparative anatomy. The brain of a lion is both bigger and heavier than that of a domestic cat, and yet we cannot say with any certainty that the intelligence of the former animal is higher in degree than that of the latter. And if we embrace in our comparison a wider field of anatomical enquiry, we can easily satisfy ourselves that amongst animals of the same genus or family, the size of the brain has a relation to the size of the species, that the smallest species have the smallest brains, and *vice versâ*. Hence we arrive at the important conclusion, that absolute differences in the size of the brain do not necessarily imply corresponding differences in the degree of intelligence; that the brain being likewise an important centre of sensori-motor activity, its size and weight in various animals bear a relation to the size and weight of the entire body; and that brain-mass and body-mass are correlated with each other.

Many observations have also been made to determine if any relations exist between the weight and size of the brain in Man and the weight and stature of the adult human

body, and between the weight of the brain and the sex or race of the individual ; and although the data are not yet sufficiently numerous to permit me to speak dogmatically on all these questions, there can be no doubt that on some precise statements may be made.

In comparing individuals with each other, we cannot say that the weight of the brain bears a direct proportion to the weight of the body. For the human brain, in all probability, attains its full size and weight at or about the age of thirty, whilst the body not only increases greatly in weight after this period, but in one and the same individual may vary considerably in weight at different stages of adult life, without any corresponding fluctuations taking place in the weight of the brain. As the body, therefore, has a tendency to increase in weight during the period of maturity, whilst that of the brain remains stationary, the relative weight of the latter during that period is always less than at or before the age of puberty.

Several observers have enquired into the relations of the weight of the brain to the stature of the individual, and by some it has been thought that the brain is on the average heavier in tall than in short persons. Amongst the Hindoos, for example, the mean brain-weight has been estimated by Dr. Barnard Davis [1] to be one-tenth less than that of the Germans or English, and in stature also they fall considerably below the general European standard. Exceptions, however, to this general proposition are by no means uncommon. Although the Laplanders are a people of low stature, the average height for the men being about five feet, their mean brain-weight, as Dr. Barnard Davis has pointed out, is equal, or almost equal, to that of a taller race, the Italians. Again, amongst people of the same race, a big head may not unfrequently be seen on a short body, and a tall man may have a head below the average size. Hence it does not seem as if any very precise conclusions can be drawn of the existence of a direct relation between stature and brain-weight.

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[1] ' Philosophical Transactions, 1868,' p. 519.

That the head of a woman is on the average smaller than that of a man is a matter of every-day observation. As the result of numerous enquiries, it has been ascertained that in all peoples and races, without any exception, the absolute weight of the entire brain is on the average always greater in men than in women, though of course individual women do sometimes possess heavier brains than individual men. Tiedemann, Sims, Clendinning, Parchappe, Reid, Bergmann, Huschke, Boyd, Peacock, Wagner, Sappey, Blosfeld, Weisbach, Thurnam, and Bischoff have weighed the brains of men and women, both sane and insane, of the German, French, and British peoples, amounting collectively to several thousand specimens. Though there are some differences amongst these observers as to what the average brain-weight in each sex may be—differences which to some extent no doubt possess an ethnic signification—yet there is a common accordance in giving a much greater brain-weight to men than to women. It has been ascertained that amongst Europeans the average weight of the brain in women is 10 per cent. less than in men ; so that if the average weight of the adult European male brain be taken as 49 or 50 ozs., that of the adult female is not more than 44 or 45 ozs. These observations are confirmed by an extensive series of brain-weighings made under the superintendence of Dr. Crichton Browne in the West Riding Lunatic Asylum [1] the average weight of 375 male brains being 49 ozs., whilst that of 300 female brains was 43·9 ozs. This difference in the two sexes is much greater than could be accounted for, even if we were to assume, as Tiedemann supposed, that a correlation existed between brain-weight and stature. From calculations founded on the elaborate Tables of Dr. Boyd, Dr. Thurnam has shown,[2] that whilst the brain-weight is nearly 10 per cent. less in the female than in the male, the stature is only 8 per cent. less. The difference, therefore, in the size and weight of the brain is obviously a fundamental sexual

[1] I am indebted to Mr. W. C. S. Clapham, one of Dr. Crichton Browne's assistants, for a copy of the table of brain-weights, from which this and succeeding calculations have been made.

[2] The Weight of the Brain, and the circumstances affecting it, in ' Journal of Mental Science,' April 1866.

distinction, and not one that can be explained on the hypo-
thesis that the educational advantages enjoyed either by the
individual man, or by the male sex generally, operating
through a long series of generations, have stimulated the
growth of the brain in the one sex more than in the other.
For important differences are found not only in the most
highly civilised peoples, where more attention may perhaps
have been paid to the development of the functions of the
brain in men than in women, but in the savage races,
where the men and women are alike uncultivated ; and
they are alike met with in the adult brain at all periods of
life, and in the newly born child. In 42 newly born male
infants examined by Dr. Boyd, the average weight of the
entire brain was 11·67 ozs. ; whilst in 39 females the average
weight was only 10 ozs. In correspondence with the greater
size and weight of the brain, the head of the male infant is
bigger than that of the female—a circumstance which, as is
well known to the obstetrician, not only retards, but even
renders more dangerous both to mother and child the process
of parturition.

Much requires to be done before we shall be in a position
to speak dogmatically of the influence which race exercises
on the weight of the brain. Amongst the cultivated Euro-
pean peoples, indeed, so many observations have been re-
corded, that there can be little doubt that the average of each
race differs but slightly from the general average of from 49
to 50 ozs. for the adult male, and about 44 ozs. for the adult
female. As yet, however, the opportunities of weighing the
brain in the uncultivated and in the savage races have
been but scanty. Drs. Reid and Peacock[1] have weighed the
brains of 7 adult African negroes, 5 males and 2 females.
In the former the mean weight of the encephalon was about
44$\frac{1}{4}$ ozs., in the latter 43$\frac{1}{2}$ ozs. Almost precisely similar
results were obtained by Tiedemann from weighing 4 male
brains, and by Barkow from 3 male brains. The average
brain-weight in the male negro, as far as these cases show,
approaches therefore that of the European female, and the

difference in the weight of the brain in the negro and negress is much less than in the two sexes amongst Europeans. The number of cases observed, more especially of negresses, is as yet too small to be used as a basis for any very decided conclusion, and we have not sufficient data before us to determine what the extreme range of variation in each sex may be. In the above specimens the heaviest male brain weighed 50·8 ozs., and the lightest 38·9 ; whilst the heavier brain of the two negresses was 46 ozs., the lighter 41 ozs.[1]

A very extensive series of observations has been made by Dr. J. Barnard Davis,[2] not on the brains themselves, but on the cubic capacities of crania, with the view of determining, after making an allowance for the membranes, blood, and cerebro-spinal fluid, the weight of the contained brain. There can be little doubt that an approximative estimate of the brain-weight may thus be obtained with a measure of accuracy ; though, as Dr. Davis admits, the estimated weights given by him somewhat exceed the true weight of the brain, and in future researches it will be desirable to make a greater deduction for the weight of the membranes and fluids.

I have abstracted from Dr. Davis's fuller and more detailed tables the following short statement of some of the most important facts elicited by his investigations, relative to the estimated weight of the brain in the two sexes, not only in the average, but in the heaviest and lightest specimens. The weights are expressed in avoirdupois ounces. The estimated average weight of 53 male African brains was 45·63 ozs. The range of variation was from 54 ozs. in a negro whose tribe was not known to 39·18 in a Congo negro. The estimated average weight of 60 female African brains was 42·74 ozs. The range of variation was from 51·23 ozs. in a Bakēlē negress to 34·15 in a negress of each of the Mandingoe and Asango tribes.

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[1] Mr. R. Garner describes in the 'British Association Reports,' Exeter Meeting, 1869, the brain of a negro, aged 61, which weighed as much as 49 ounces. Dr. Kopernicki, again, has described the brain of a negro, aged 35, which weighed only 1105 grammes = 38·97 ozs. ('Revue d'Anthropologie,' Paris, 1872, p. 121).

[2] 'Philosophical Transactions, 1868,' p. 505.

The estimated average brain-weight of 24 males of the Australian races was 42·83 ozs. The range of variation was from 53·35 ozs. to 36·71. The estimated average brain-weight of 11 females was 39·22 ozs. The range of variation was from 44·08 ozs. to 34·15.

The estimated average brain-weight of 210 males of the Oceanic races was 46·54 ozs. The range of variation was from 56·48 ozs. in a Maduran islander to 37·22 in a Dayak. The estimated average brain-weight of 95 females was 43 ozs. The range of variation was from 52·76 ozs. in a Kanaka to 37·58 in an Amboynese and in a Malay.

The estimated average brain-weight of 299 male Europeans was 48·25 ozs. The range of variation was from 63·49 ozs. in an ancient Romano-Briton to 38·82 in an ancient Scottish. The estimated average brain-weight of 94 females was 42·49 ozs. The range of variation was from 52·54 ozs. in an Irish to 31·67 in a Romano-British skull. The weight of the last-named specimen was so peculiarly and exceptionally small, that it can scarcely be regarded as a normal brain. In none of the other female European skulls did the brain-weight fall below 36 ozs.

The conclusions which one may legitimately draw from an analysis of these tables are, 1st, that the average brain-weight is considerably higher in the civilised European than in the savage races ; 2nd, that the range of variation is much greater in the former than in the latter ; 3rd, that there is an absence or almost complete absence of specimens heavier than 54 ozs. in the exotic races, so that the higher terms of the series are not represented ; 4th, that though the male brains are heavier than the female, there is not the same amount of difference in the average brain-weight between the two sexes in the uncultivated as in the cultivated peoples.

These conclusions are, as regards the English people, borne out by Dr. Boyd's observations on 2,086 brains, said to be from sane persons, [1] examined in the St. Marylebone In-

[1] 'Philosophical Transactions, 1861.' Although by far the greater number of these cases were undoubtedly from sane persons, yet as many as 17 per cent. had died of diseases of the nervous system, including the brain. A few were idiots,

firmary.   Between the ages of 14 and 70 he found the male
brain to range in weight from 60·75 to 30·5 ozs., and the
female from 55·25 to as low as 27·5 ozs. :· these lowest weights
are, however, exceptional ; and the next lowest adult male
brain recorded by Dr. Boyd is 33·75 ozs., and adult female
32·5 ozs.[1]   As a still further illustration of the weight which
the normal brain may attain, I may state that in the Ana-
tomical Museum of the University of Edinburgh is the brain
of a boy, æt. 15, who died after the operation of lithotomy,
in whom the brain weighed 60 ozs. ; and that Dr. Peacock
met with four male brains in the Pathological Theatre of
the Edinburgh Royal Infirmary, which ranged in weight
from 61 to 62·75 ozs.   In none of these individuals was there
any evidence of the possession of superior intellectual ca-
pacity.

But in discussing the size and weight of the entire brain,
in relation to the intellectual power of the individual or of
the race, it must be kept in mind that the gross weight of
the organ represents much more than the weight of the
cerebrum, and includes that of the optic lobes, the pons,
cerebellum, and medulla, which serve as important sensori-
motor organs, not directly associated with intellectual
activity.   And further, that the weight of the cerebrum
itself represents not only that of the convolutions, but of the
optic thalami and corpora striata.

No reliable determinations have as yet been made of the
exact proportion, as regards bulk and weight, which the
convolutions bear to the internal ganglia ; and the anatomi-
cal relations, indeed, of the one to the other are of such a
kind as to make it extremely difficult to estimate them.
Data are, however, at our disposal to enable us to form an
estimate of the relative weight of the pons, cerebellum, and
medulla to the entire encephalon.   From Dr. Boyd's Tables

and it is possible that the brains of low weight referred to in the succeeding sen-
tence in the text were from cases of idiocy or imbecility.

[1] Dr. Boyd's weighings were not made on the entire brain, but after it had been
sliced.   The weights, therefore, whilst apparently including the membranes, do not
include the fluids of the brain, which would necessarily to a large extent have
drained away.

of the brain-weights of sane persons it can be shown that in about 500 males, between 20 and 70 years of age, the relative weight of these parts to the entire brain is 12·97 per cent., and in an almost equal number of female brains 12·93 per cent. From the Wilts County Asylum Tables of the brains of the insane, compiled by Dr. Thurnam,[1] we can calculate the proportion as 12·9 per cent. in the male, and 13·1 in the female, during the same periods of life ; and the West Riding Asylum Tables show an almost similar proportion—viz. 12·8 per cent. in 237 male brains, and 13·0 in 262 females. In round numbers, therefore, we may say that the pons, cerebellum, and medulla, between the ages of 20 and 70, bear to the entire encephalon the ratio of 13 to 100, and that this relative weight is practically the same in the two sexes. Hence the smaller weight of the brain in women than in men is due, not to a greater proportional reduction in any one part of the organ but to a diminution in which all its subdivisions participate.

But though these general conclusions may be drawn as to the average proportion to be deduced from the weighings of a large number of brains, yet there can be little doubt that in individual brains a departure from this average ratio may and does arise. A brain exceeding in size and weight the average of the race may in some cases owe its increased bulk, not to any special amplification of the convolutions, but to a greater proportional increase in the other subdivisions of the encephalon. Whilst, conversely, a brain somewhat below the average may yet have its convolutionary area developed in a greater ratio than the other parts.

Again, from the data at our disposal, it seems certain, if the human brain, even amongst the most uncultivated peoples, falls below 30 ozs., that this low weight is not merely incompatible with intellectual power and activity, but is invariably associated with idiocy or imbecility ; so that the human brain has a minimum weight below which intellectual action is impossible. Amongst the more cultivated peoples the minimum weight-limit of intelligence is, however, in all probability higher than 30 ozs. It has been placed by M. Broca

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[1] Op. cit. Table I.

at 32 ozs. for the female and 37 ozs. for the male brain, and Dr. Thurnam's numbers are almost the same. To how low a weight the brain in the microcephalous idiot may fall is well shown in a case recorded by Theile, where it weighed only 10·6 ozs., in Mr. Gore's case of 10 ozs. 5 grs., and in Mr. Marshall's case of 8½ ozs.[1]

But it by no means follows, when a brain exceeds this minimum term, that it should be a competent organ of thought. The tables of the weights of the brain in the insane which have been published by Dr. Boyd, M. Parchappe, and Dr. Thurnam, furnish many examples of brains which weighed 55 ozs. and upwards. Dr. Bucknill met with a brain in a male epileptic, æt. 37, which weighed 64·5 ozs., and Dr. Skae observed in a female monomaniac, æt. 39, a brain which weighed 61·5 ozs. In the West Riding Asylum Tables also, of 375 males examined, the weight of the brain in 30 cases was 55 ozs. or upwards ; and the particular forms of insanity exhibited were melancholia, mania with and without epilepsy, general paralysis, and dementia. The highest weights were 61 ozs. in a case of senile dementia, 60½ ozs. in a case of dementia, and 60 ozs. in one of melancholia. Of 300 females examined, the weight of the brain in 26 cases was 50 ozs. or upwards, and the forms of insanity exhibited were melancholia, mania, dementia with and without epilepsy, and in one instance imbecility. The heighest weights were 56 and 55 ozs. in two cases of mania. In the same tables the weight of the brain in 7 male and 3 female idiots is recorded, and in no instance is the brain-weight less than 34 ozs., and, as may be seen from the accompanying table, in 5 cases exceeded 40 ozs. :—

Sex	Age	Brain-weight	Sex	Age	Brain-weight
		Ozs.			Ozs.
M.	31	36	F.	16	36
M.	17	47¾	F.	17	42
M.	39	36½	F.	18	34
M.	16	43			
M.	10	44			
M.	22	46			.
M.	19	34			.

[1] 'Philosophical Transactions, 1863,' p. 527.

But instances are not wanting in which the brains of idiots have exceeded even 50 ozs.   One very remarkable example has been communicated by Dr. Langdon Down to Dr. Thurnam, in which the brain of a male idiot, aged 22, weighed 59·5 ozs. ; and Dr. J. Batty Tuke has recently met with the case of a male idiot æt. 37 where the brain weighed 60 ozs., and the cranium, which was fully ossified, had an internal capacity of $110\frac{1}{2}$ cubic inches.[1]

From these and the other facts previously considered, it is clear that the size and weight of the brain cannot *per se* give an exact method of estimating the intellectual power of the individual, and that a high brain-weight and great intellectual capacity are not necessarily correlated with each other.

EXTERNAL CONFIGURATION.—In the human brain, and in the higher mammalia generally, the surface of the hemispheres presents a more or less folded appearance, technically called the convolutions or gyri.   These convolutions are not, as was at one time supposed, irregular and inconstant in their disposition, but possess, as was first systematically described by Gratiolet,[2] definite modes of arrangement.

The human brain, for example, can be subdivided into five lobes by certain fissures seen on the surface of the hemispheres, which lobes have been called temporo-sphenoidal, occipital, frontal, parietal, and the insula or central lobe.   The convolutions of the temporo-sphenoidal lobe are arranged in three parallel tiers from above downwards, and are separated from the fronto-parietal lobes by the well-known Sylvian fissure.   The occipital lobe, small in size, forms the hinder end of the cerebrum, and also possesses, from above downwards, three parallel tiers of convolutions.   It is separated from the parietal lobe by the parieto-occipital fissure, which fissure, on the outer surface of the hemisphere at least, is bridged across by two convolutions, named by Gratiolet *plis de passage*, and by British anatomists the first and second bridging or annectent gyri.

[1] 'Journal of Anatomy and Physiology,' May 1873.
[2] 'Mémoire sur les Plis cérébraux de l'Homme et des Primates.'   Paris, 1854.

The great fronto-parietal lobes are subdivided into frontal and parietal portions by a fissure which runs obliquely upwards and backwards over the outer face of the hemisphere, and is named the fissure of Rolando. The frontal lobe consists of a large convolution, which ascends immediately in front of the fissure of Rolando ; of three tiers of convolutions, named superior, middle, and inferior frontal convolutions, extending from the anterior end of the cerebrum backwards, and parallel to each other to join the ascending frontal convolution; and of some small convolutions on its orbital surface. The parietal lobe consists of a large convolution which ascends immediately behind the fissure of Rolando, and of three convolutions, named postero-parietal lobule, supra-marginal convolution, and angular convolution, which pass backwards to the parieto-occipital fissure and the posterior extremity of the fissure of Sylvius. The insula, or Island of Reil, is composed of from five to seven short gyri which radiate outwards and backwards from the locus perforatus anticus, and lie concealed within the Sylvian fissure. On the inner face of the hemisphere also a well-defined and long-recognised convolution, the gyrus fornicatus, is seen winding round the corpus callosum ; to its posterior and lower end the name of gyrus hippocampi is now not unfrequently applied. Other less extensive, but at the same time well defined, convolutions may also be seen on this inner surface.

We may now not unreasonably ask if this mode of arrangement of the convolutions of the human cerebrum is absolutely distinctive ? Is this plan so entirely different from that found in other mammals that we might say, here is something so characteristic of the brain of man that it is obviously correlated with his intellectual pre-eminence ?

If we, for example, compare the human brain with that of a rabbit or other rodent, the absence of convolutions in the latter at once shows us a most important difference in the external configuration of the cerebrum. In the carnivorous, pachydermatous, and ruminant mammals, although the brain is convoluted, yet the convolutions are not arranged in definite lobes as in the human brain, but lie in successive tiers

around the Sylvian fissure, and the number and distinctness of these tiers are by no means uniform in different genera.

In the brain of the spider-monkey, or, still better, that of the macacus, a subdivision into lobes and a foreshadowing of convolutions arranged on the human plan may be recognised. But in the anthropoid apes, as the orang and chimpanzee, the method of arrangement closely approaches that seen in the human brain; for not only are the five lobes very distinct, but similar primary convolutions are also present. And although the brains of the anthropoid apes are not so richly provided with the smaller secondary or tertiary convolutions as is that of man, yet these annectent or bridging gyri are by no means so absent in the former as was at one time supposed, and variations in the size, number, and disposition of these gyri may occur in the ape's brain as in the brains of men.[1] But even if it had been universally true that these annectent gyri were constantly present in man, and as constantly absent in the ape, such minor morphological differences would clearly have been insufficient to have bridged over the enormous gap which distinguishes the intellects of the authors of the Principia and of Hamlet from the intelligence, whatever it may be, displayed by the jabbering, tree-dwelling orang and chimpanzee.

But important differences exist, both in size and weight, between the human brain and that of the ape. Professor Owen found the brains of a half-grown male, and of a female chimpanzee to weigh, the one $9\frac{3}{4}$ ozs., the other $13\frac{1}{4}$ ozs.;[2] Mr. Marshall found the brain of a young male chimpanzee to weigh, along with its membranes, 15 ozs.[3] The brain of a young male orang, examined by Professor Rolleston,[4] weighed 12 ozs. These weights are far below those of the adult human brain, and do no more than approximate to the

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[1] See, for example, Dr. Rolleston's description of the brain of the Orang in 'Natural History Review,' vol. i. p. 201, and his Lecture in 'Medical Times and Gazette,' 22nd February and 15th March, 1862; also my 'Notes on the Bridging Convolutions in the Brain of the Chimpanzee' ('Proc. Roy. Soc. Edinburgh, 1865–66'), and my pamphlet 'On the Convolutions of the Human Cerebrum, 1866;' also M. Paul Broca's Memoir, 'L'Ordre des Primates,' Paris, 1870, p. 162.

[2] 'Trans. Zool. Soc.,' vol. i. p. 343.

[3] 'Natural History Review,' vol. i. p. 297.   [4] Ibid. p. 207.

average weight in the human fœtus at the time of birth.[1] These differences in size and weight not only affect its entire bulk, but the volume of the individual convolutions, which in the ape are very much smaller than in the adult man. For the purposes of ape-life, however, the low brain-weight and the small convolutions are sufficient to enable the animal to perform every function of which it is capable. Its muscular and nervous systems are so perfectly co-ordinated that it can move actively about from tree to tree, can seize and retain objects with the greatest precision, can search for and obtain its food, and even can build a habitation for its residence. In all these respects it presents a striking contrast to the young infant, with an almost similar brain-weight, and with equally small convolutions, lying helpless on its mother's knee.

Few opportunities have as yet been obtained of comparing the size and complexity of arrangement of the convolutions in the savage with the cultivated races of men. Two ana-tomists, however, have been so fortunate as to acquire and examine specimens of the brains of women of the Bush race, one of the most primitive of savage peoples. From the care-ful descriptions of M. Gratiolet [2] and Mr. Marshall,[3] it is clear that in these specimens the arrangement of the convo-lutions was less complicated than in the average European brains—the greater simplicity being due to the paucity of the secondary or tertiary gyri connecting the primary convo-lutions together, and to the less tortuous disposition of the convolutions generally. The brain-weight of M. Gratiolet's specimen does not seem to have been recorded. Mr. Mar-shall estimates that of his specimen to be 31·5 ozs. The brain-weight of a Bosjes girl, æt. 14, seen by Dr. R. Quain,[4] is stated to have been 34 ozs.; and in another Bosjes girl,

[1] Some years ago (' Proc. Roy. Soc. Edinburgh,' 16th January, 1865) I measured the cubic capacity of some crania both of the chimpanzee and the gorilla, which were as follows :—Adult male gorilla 28 cubic inches ; adult female 26·5 ; young female 23 ; adult male chimpanzee 22 cubic inches ; adult female 24 ; younger specimen 21. These capacities are very much below that of the human skull.

[2] ' Snr les Plis cérébraux de l'Homme et des Primates.' Paris, 1854.

[3] ' Philosophical Transactions, 1863.'

[4] ' Pathological Transactions, 1850,' p. 182.

whose brain was removed by Messrs. Flower and Murie,[1] the weight, when deprived of the greater part of the membranes, was 38 ozs. Thus the comparatively simple arrangement of convolutions is accompanied by a brain-weight considerably below the European average.

To some extent data have now been collected, which show that some men of high intellectual capacity have possessed brains the convolutions of which were complex in arrangement from the increased development of the secondary and tertiary gyri and from the greater tortuosity of the convolutions generally. The brains of the eminent mathematicians Gauss and Dirichlet, so beautifully figured by Rudolph Wagner, are illustrative examples.[2] Careful measurements of the convolutions conducted by Hermann Wagner have shown that the superficial area of the convolutions in the brains of Gauss and of Professor Fuchs was considerably greater than in an ordinary man or woman. But, as Wagner has himself pointed out, great intellectual activity is not of necessity limited to brains which possess great complexity in the arrangement of the convolutions ; and he adduces, in . illustration of this fact, Hermann the philologist and Hausmann the mineralogist, in neither of whom were the brainweights above the average, nor the convolutions arranged in a complex manner.

The convolutions may to a considerable extent vary in size in the human brain without their functional activity being appreciably affected ; though we do not possess the same numerical data to determine what the minimum size may be which is compatible with intellectual vigour as we do in the case of the minimum weight. This, however, we do know, that when reduced to a state of tenuity from atrophy or congenital mal-development, serious mental defects are occasioned.[3] On the other hand, it by no means

[1] ' Journal of Anatomy and Physiology,' vol. i. p. 206.

[2] ' Vorstudien.' Göttingen, 1860, 1862.

[3] A case in which the condition of the patient during life, and the anatomical characters of the brain as revealed in the *post-mortem* examination, are very carefully described, is recorded by Dr. Kenneth M'Leod in the ' Edinburgh Medical Journal, October 1864.' The brain is preserved in the Anatomical Museum of the University of Edinburgh. Cases illustrating the co-existence of structural defects in the

follows, when the convolutions are normal in size and shape
and relative position, that they should be perfect in func-
tion.   For not only may we find the convolutions of the
brains of the insane well formed and normally arranged,
but even in idiots and imbeciles, where there is a positive
absence of intellectual power—though this serious defect
is not unfrequently associated with some grave error in
development—the convolutions have in many cases possessed
their normal form and arrangement.

Hence it is clear that a correspondence in morphological
configuration by no means necessitates either equality or
similarity in functional power.   In estimating the value of
the convolutions, therefore, either when the brains of men
are compared with each other, or with those of animals,
other factors are to be considered than those afforded by size,
or weight, or form, or modes of arrangement.

INTERNAL STRUCTURE.—The grey colour of the surface of
the convolutions of the cerebrum is familiar to everyone who
has seen a brain when the membranes have been stripped
off.   When a convolution is bisected, the grey matter is
observed to be laminated, the layers being arranged parallel
to the plane of the surface, and in the axis of the convolution
is a white core, the strands of which radiate into the
surrounding grey matter.   When thin slices through the
white core are examined microscopically, it is found to
consist of nerve fibres, and blood-vessels, embedded in a very
delicate, richly nucleated protoplasm, which seems to be an
imperfectly differentiated form of connective tissue, and by
binding the other constituents together has been named by
Virchow the Neuroglia or Nerve-glue.   The grey cortex
contains similar constituents, but possesses, in addition,
numerous cells, which are its especial anatomical character-
istics.   It is generally admitted that the nerve fibres,
whether they occupy the white core or the grey cortex, are
conducting rods, protected by an insulating investment, the
white substance of Schwann, just as the copper wires in a

brain with imperfection in the senses and intelligence, are recorded by Dr. Broad-
bent in the 'Journal of Anatomy and Physiology,' vol. iv. p. 218; and by Mr.
Messenger Bradley in the same Journal, vol. vi. p. 65.

telegraph cable are surrounded by insulating substances; whilst the characteristic cells of the grey cortex are the structures in which the molecular changes occur, which occasion the evolution or disengagement of the special form of energy named nerve-energy or nerve-force.

It becomes, therefore, an important object of enquiry to determine the extent and thickness of the grey cortex in the different parts of the same brain, as well as in different brains,[1] in order that an approximative estimate may be formed of the value of the convolutions as centres or fountains of origin of the special modification of energy which is evolved by them—I say, only an approximative estimate, because it must not be forgotten that the grey matter contains a proportion of neuroglia, which, inoperative as regards nerve-work, acts only as a packing material, and to the nerve-elements bears a relation not unlike the dull yet space-filling 'padding' intermingled with the thoughtful and brilliant writings of, say a Carlyle or a Macaulay, in some magazine, where quantity and not quality merely have to be provided for the reading and subscribing public. There can be no doubt that in some brains, in which an unusual thickness and superficial area of the convolutions are met with, they owe their increased relative amount not to a greater development of the nerve-elements, but of the neuroglia.[2]

To arrive, therefore, at a proper conception of the value of the grey matter, the study of its minute structure as displayed by the microscope should proceed *pari passu* with the observations on its thickness, so as to determine the size, relative numbers, arrangement, and connections of the nerve cells and fibres in any given area, and in the entire brain.

And here we have opened up to us a wide field of anatomical enquiry—one which includes not only the comparison of the minute structure of the grey matter of the different convolutions of the same human brain, but of various human

[1] An ingenious little instrument named a Tephrylometer has recently been invented for this purpose by Dr. H. C. Major, and described by him in the last part of the 'West Riding Asylum Reports,' p. 157.

[2] An illustrative case in confirmation of the above statement has just been recorded by Dr. J. Batty Tuke in the 'Journal of Anatomy and Physiology,' May 1873.

brains with each other, and these again with the brains of various animals.   And it is fortunate for present and future investigators that the difficulties connected with this line of research have, up to a recent period, been so great as to leave comparatively untrodden   ground.   With   the   improved methods of observation which have of late years been introduced, and with the still further improvements which may reasonably be looked for, any competent observer who will devote his time to working at this subject will find a rich reward for his labour in the new facts which he will acquire.

But we are not altogether ignorant of certain facts appertaining to the nerve cells of the convolutions of the human brain.   By the published observations of Lockhart Clarke,[1] Arndt,[2] Cleland,[3] and Meynert,[4] and from a number of specimens which I have myself examined, there   can   be no doubt that these cells vary in relative size, in numbers, and in shape in the different layers of the grey cortex.   The greater number of the cells, however, exhibit one characteristic form, which has been called pyramidal, the basal   part of   the cells being   directed towards the white core, whilst their apices extend towards the surface of the convolution in the form of remarkably elongated processes, which, according to Cleland, are   continued   into   the   nerve   fibres   forming a   horizontal layer on the surface of the convolutions.   Very fine processes arise from these cells, but   one   proceeds from the   base of each, which, as Koschennikoff's [5] observation shows, is continuous with one of the vertical or radiating nerve fibres of the white core.   Others of the cells have a fusiform shape ; but of these I have also seen one of the processes prolonged towards the surface of the   convolution, the   other   towards the white core ; others of the cells, again, have the form of stellate nerve corpuscles.

The largest-sized pyramidal cells lie   in   the   deeper layers

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[1] ' Proc. Roy. Soc.' Lond. 1863 ; and Maudsley's ' Physiology and Pathology of Mind,' 1868.

[2] Schultze's ' Archiv,' 1867, and subsequent years.

[3] ' Quart. Microscopic Journal,' 1870.

[4] ' Vom Gehirne der Säugethiere ' in Stricker's ' Handbuch der Lehre von den Geweben,' 1870.

[5] Schultze's ' Archiv,' vol. v. p. 374.   See also the newer observations of Butzke in ' Archiv f. Psychiatrie,' 1872; and of Gerlach and Rindfleisch in 'Centralblatt,' 1872.

of the grey matter. Some years ago it was stated by Lockhart Clarke that the cells of all the layers of the posterior or occipital lobe were small and of nearly uniform size, whilst in the convolutions in front of this lobe numerous cells of a much larger kind were found. It is undoubtedly true that large pyramidal cells are found in the frontal lobe in considerable numbers, and that the greater number of the cells of the occipital lobe are small, and nearly uniform in size, but it is not correct to say that no large pyramidal cells occur in the convolutions of the latter lobe. Meynert, for example, has described a few pyramidal cells of very large size in this region ; and in a well-prepared section through the occipital lobe of a perfectly healthy brain, prepared and presented to me by Dr. Major, there is no difficulty in recognising a small proportion of cells quite equal in size to the largest cells of the frontal lobe, interspersed amongst the smaller pyramidal cells.

There can be little doubt that the larger nerve cells are functionally more active, and evolve more nerve energy than do those of smaller size. Though more extended data in support of the anatomical continuity of the nerve cells of the convolutions with the nerve fibres is to be desired, yet on physiological grounds there can be little doubt that each nerve cell has at least one nerve fibre, either directly or indirectly continuous with it, so that multitudes of conducting rods are provided in the large nerve centres, along which the energy evolved by the nerve cells can be transmitted. It would be very difficult indeed to conceive how nerve fibres could fulfil the functions which are universally ascribed to them, if they were not anatomically continuous with nerve cells, as without such connection they would be like strings of knotless threads, or the wires of a telegraph cable dissevered from their central battery.

In studying the arrangement of the nerve fibres of the cerebrum, it is of great importance to determine the portions of the cerebrum, and the groups of nerve cells with which they are continuous, as without this knowledge it is not easy to determine what the parts are which are associated together. And here, just as in the study of the nerve cells, though we

know some of the broader and more general facts, we are
much in need of fuller and more precise information.

We know, for example, that a great system of longitudinal
or peduncular fibres ascends from the spinal cord and
medulla oblongata through the pons and crura cerebri to
the optic thalami and corpora striata ; from which the fibres
may either directly, or indirectly after junction with the
nerve cells of those bodies, radiate outwards to the grey
cortex of the cerebrum, more especially to that of the frontal
lobes.   But peduncular fibres also ascend from the grey
matter of the spinal cord and medulla to the cerebellum,
and others again, arising in the cerebellum, ascend to the
corpora quadrigemina, which latter again are connected
with the cerebrum proper by peduncular fibres.

We know also that a great system of transverse fibres
passes across the mesial plane from one hemisphere to the
other.   The most numerous and important of these fibres
constitute the corpus callosum, which connects together
corresponding couvolutions in the opposite hemispheres, and
its fibres decussate with the fibres of the peduncular system,
passing to the same convolutions.   But smaller groups of
fibres also cross the mesial plane.   The anterior commissure,
for example, as was pointed out by Spurzheim half a century
ago, passes between and connects together the convolutions
of the opposite temporo-sphenoidal lobes, and in its course
traverses the two corpora striata, which, in all probability, it
also brings into anatomical connection with each other.   The
posterior commissure, again, connects the two optic thalami.

But in each hemisphere, also, there are numerous fibres,
collected together into more or less distinct bands, which,
under various descriptive names, as fornix, tænia semicircu-
laris, nerves of Lancisi, fasciculus uncinatus, &c., run in the
antero-posterior direction for a greater or less distance, as
the case may be, to connect together structures situated on
the same side of the mesial plane.   Amongst the most
important of these fibres are those which connect together
adjacent convolutions, or groups of convolutions.

Lastly, from the grey matter in certain of the subdivisions
of the encephalon bands of nerve fibres arise, which, under

the name of cranial nerves, connect the brain with various sensory and motor peripheral end organs. But of the cranial nerves only two, viz. the olfactory and optic, appear to have any immediate connection with the grey matter of the convolutions, all the rest taking their origin in groups of nerve cells, forming the centres which lie nearer the base of the encephalon.

From these general facts, we reach the very important conclusion, that the convolutionary area of the hemispheres does not form a system dissociated from the other nerve centres ; that not only are convolutions in the same hemisphere and in opposite hemispheres connected together, but that they are anatomically continuous with the various centres from which the cerebro-spinal nerves arise, and through these are brought into relation with the outer world.

Vascular Supply.—It is a well-known axiom in physiology that organs which display great functional activity require and receive an abundant supply of blood, so that the work performed by the organ bears a relation to the amount of blood which flows through it in a given time. The brain ranks as one of the most vascular organs, for not only does it receive its blood through four good-sized arteries, the vertebrals and internal carotids, but these vessels communicate so freely with each other within the cranial cavity as to permit of a ready transmission of blood to the different parts of the organ. And it is interesting to observe, in connection with the distribution of the blood to the substance of the brain, that by far the greater number of the blood-vessels penetrate the convolutions in their passage to the deeper parts. As they go through the grey matter they give rise to a very complete network of capillaries, which have been computed to be five times more abundant in the grey cortex than in the white core of the convolutions, a circumstance which substantiates the view that the grey matter is functionally much more active than the white.

The amount of blood which flows to a given brain, relatively to its size, is a point that ought to be kept in view not only in connection with its own activity, but in comparing it

with other brains.   An illustration of my meaning may be
drawn from comparative anatomy.   It is well known that the
Cetacea possess brains of considerable size, and that, with
the exception of the elephant, the larger whales are the only
animals which possess brains absolutely bigger than those of
man. It is stated, for example, that Dr. Scoresby ascertained
the weight of the brain in the Greenland whale, *Balæna mys-
ticetus*, to be 3 lbs. 12 ozs. Herbert Spencer,[1] in considering
the reason why the brain of a porpoise should exceed in size
the brains of most other mammals that have bodies com-
mensurate with its own, thinks that its great size of brain
is correlated with the expenditure of energy needed to keep
up the high rate of speed with which it moves through
the dense liquid medium in which it lives.

Some years ago, when engaged in the dissection of the
sucking calf (about 8 feet long) of a pilot whale, *Globio-
cephalus melas*,[2] I found that the brain, even after having
been immersed for some weeks in spirits of wine, and there-
fore several ounces below its normal weight, weighed as
much as 58 ozs. avoirdupois.   I also observed that the in-
ternal carotid arteries, in their passage upwards to the brain,
became so much reduced in size that the quantity of blood
which they could have conveyed to it was much smaller than
could be transmitted by the corresponding arteries in man.
It is interesting to note that, some years before I made this
observation, Dr. Sharpey[3] and Von Baer[4] had directed atten-
tion to the great diminution in size of the internal carotid
arteries of the porpoise before they enter the cranium.
Hence it seems to me not improbable that the brains of the
Cetacea, relatively to the size of these animals, are function-
ally not so active as those of the smaller and more highly
vascular brained mammals ; so that, to perform the work
which they are called upon to do, an absolutely greater mass
of brain substance is needed.

But the arteries of the brain, as elsewhere, are not merely
to be regarded as tubes with simple elastic walls capable of

[1] 'Principles of Psychology,' 2nd edit. 1870, p. 8.

[2] 'Journal of Anatomy and Physiology,' vol. 2, p. 69.

[3] 'Reports of British Association,' 1834, p. 683.

[4] 'Nova Acta Phys. Med.,' vol. xvii. 1835.

dilating under the force of the blood pressure due to the heart's contraction, and then recoiling when that contraction has ceased; but as pipes which possess in their own walls a musculo-nervous apparatus, which accurately regulates the amount of blood transmitted through them in accordance with the nutritive needs of the parts.   The importance of studying the connections and arrangement of this vaso-motor apparatus, in its relations to the physiological and pathological changes in the various arterial cerebrospinal areas, has been pointed out by my colleague, Professor Laycock, in a very suggestive lecture recently [1] published in the ' Medical Times and Gazette.'

After the survey, necessarily condensed from the limited space at my disposal, of the more important facts appertaining to the arrangement and structure of the convolutions, I may now briefly indicate the purpose which is served by the convoluted surface.

If we compare the smooth brain of a rabbit, or of a marmoset monkey, with a convoluted brain, we at once recognise that in a given bulk the foldings of the latter bestow on it a far greater superficial area of grey matter than is possessed by the smooth-surfaced brain of the former; and, consequently, supposing the grey matter to be of uniform thickness in the two brains, a larger proportion of nerve cells would be found in the one brain than in the other.   But further, this differentiation of the surface into definite convolutions gives a precision to the whole method of arrangement, maps it out into more formal and easily recognisable areas, than is possible in a smooth unbroken surface, and enables us to bestow on any given group of nerve cells a local habitation and a name.

This precision of form naturally leads one to the consideration of the oft-mooted question, Are the convolutions distinct organs, each endowed with properties peculiar to and characteristic of itself?   It would be quite out of place in this Essay to attempt any analysis of the composition of mind, or any classification of its primitive or elementary

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[1] August 19 and September 2, 1871.

faculties.   But it may not be without interest to pass in
review the general character of the evidence, which has been
adduced in support of the proposition that particular facul-
ties, or properties, are localised in special convolutions, or
groups of convolutions, as well as certain difficulties which
stand in the way of too absolutely accepting this proposition.

It is universally admitted that the cerebro-spinal nervous
axis is subdivided into several regions, as the cord, the
medulla oblongata, the cerebellum and pons, the sensori-
motor ganglia, the cerebral convolutions, each of which,
though consisting both of grey and white matter, is distin-
guished from the rest by peculiarities both in outside form
and internal structure, and the physiological differentiation
of which has been abundantly proved by experiment.

Gall and Spurzheim, and the other writers of their school
of Phrenology, also claim to have been able to subdivide the
convolutionary surface of the hemisphere into areas, or
organs, each of which is the seat of a particular faculty.
But though Spurzheim had undoubtedly recognised[1] a re-
markable regularity in the general form and direction of the
convolutions even of the human brain, and though Reil,
Rolando, Foville, Huschke, and other anatomists had also
given special descriptions of certain convolutions, yet it was
not until the appearance of Gratiolet's great work[2] that the
surface of the cerebrum was accurately mapped out, and the
individual convolutions designated by definite descriptive
names.   Hence, it is only since the topography of the con-
volutions has been more precisely understood that we have
been in a position to institute a strictly scientific inquiry
into their organology.   Can we, then, find amongst these
convolutions such structural and functional differences as
would justify us in regarding them to be distinct organs?
The determination of this question, which involves most
complex problems, can only be arrived at by a patient and
prolonged combination of the three lines of biological re-
search, the pathological, the physiological, and the anatomi-

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[1] 'The Anatomy of the Brain,' translated by R. Willis, London, 1826, p. 111.

[2] Op. cit. 1854.

cal, and is not to be settled by hasty attempts at generalisation from a few empirically observed facts.

The importance of the pathological line of research has been amply illustrated by the enquiries of Broca, and various subsequent writers, into the co-existence of lesion of the posterior third of the inferior left frontal convolution, with loss of the cerebral faculty of speech. Alienists and clinical physicians are now accumulating facts, which, in the course of time, will enable this and other kindred questions to be settled.

The physiological line of research is one which, as regards its application to the convolutions of the human cerebrum, presents many difficulties, some of which, from the impossibility of applying experimental methods to the living human brain, are insuperable. Attempts have recently been made by experimenting on the brains of mammals, both on those which possess smooth surfaces, as the rabbit, and those which, like the cat and dog, possess distinct convolutions, to arrive at definite conclusions. Fritsch and Hitzig,[1] by passing electrical currents through various parts of the brains of dogs, have come to the conclusion not only that a large part— namely, the anterior—of the convex surface of the cerebrum is concerned in motor excitation, but that the posterior part is not motor ; further, that single psychical functions, and probably all, are related to circumscribed centres of the cortex of the cerebrum. This line of research, with some important modifications in the method of experimenting, is now being followed out by Dr. Ferrier,[2] who has obtained results which he regards as supporting the view that the anterior portions of the cerebral hemispheres are the chief centres of voluntary motion and the active outward manifestation of intelligence. The relations of the frontal lobes to the voluntary movements might indeed have been predicated from the anatomical connections which are known to exist between

[1] Reichert and Du Bois-Reymond's ' Archiv,' 1870, p. 300. Hitzig has a separate paper on the effects produced by galvanisation of the brain, in the same Archiv for 1871.

[2] An abstract of his results is printed in ' British Medical Journal,' April 26, 1873. The detailed research, which I have not seen, appears in the present number of the ' West Riding Asylum Reports.'

these lobes and the great motor corpora striata.   Ferrier,
moreover, supports the doctrine that the individual con-
volutions are separate and distinct centres.

Since Fritsch and Hitzig's paper was published, Professor
Nothnagel of Freiburg has communicated [1] the results of a
series of experiments on the physiology of the brain, per-
formed by injecting minute quantities of a concentrated
solution of chromic acid into definite portions of the cerebral
substance.   He urges, as resulting from his experiments,
various objections to the view that the psychical functions
are strictly localised in definite centres of the cerebral
hemispheres, and supports the older view of Flourens and
other experimenters, that these functions are bound up in
the collective mass of the hemispheres.

In connection with these methods of experimentation,
there are various disturbing causes, the effects of which
require to be very carefully considered before satisfactory
conclusions can be drawn.   Great difficulties lie in the way
of establishing that the irritation is conducted by direct paths
exclusively along definite strands of nerve fibres from the
point irritated to the regions where effects are observed.
Unless the greatest care be taken to localise its effects, the
galvanic current would, for example, at the same time be
diffused by the conducting property of the brain tissue gene-
rally to a greater or less distance around the point of irrita-
tion.   Should the latter take place, then the effects produced
would be due to a complication of secondary and reflected
nerve actions with the primary action of the irritant, and in
the analysis of these effects, the action of the one would have
to be eliminated from that of the other.   But further, the
results of experiments even on the convoluted brains of cats
and dogs, and still more on the smooth-surfaced brains of
rabbits, cannot without considerable qualification be made
to apply to the more highly convoluted human brain, until
the morphological identity of the various areas on the sur-
face of the hemispheres has been established.   And even if
we were in a position to state definitely the areas on these
different brains which are homologous with each other, al-

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[1] 'Centralblatt,' No. 45, 1872 ; and Virchow's ' Archiv,' lvii. 184, 1873.

though centres either of motion or sensation might, from the visible effects produced, be readily determined, yet, in the indefinite state of our knowledge of the psychical functions of these animals, the localisation of definite mental faculties in the individual convolutions of the human brain, as a deduction from experiments performed on the lower mammals, would be very hazardous.

If we pass now to the anatomical method of enquiry, we at once recognise that as regards outside form the convolutions possess a regularity, both in position and arrangement, which is characteristic of different genera, and even species.   Is specialisation of form, then, of itself a sufficient test of special-isation of function?   Assuredly not!   For if we examine other organs, as the kidney, the liver, or the lung, and study them in various groups of animals, we find them subdivided into distinct lobes, each of which, instead of possessing some special and distinctive property, performs precisely the same function as its neighbours.

But though the convolutions exhibit special forms, are they so individualised as to be disconnected from each other? To this question also we must answer, No!   For the grey matter on the surface of one convolution forms invariably a continuous layer with the grey matter on the surface of all the convolutions which immediately surround it, so that, if one of these convoluted brains were to be unfolded, it would present a uniform and continuous surface of grey matter similar to that of a naturally smooth-surfaced brain.   And in this respect a group of cerebral convolutions differs from a group of muscles, for in the latter each muscle is isolated from those which lie next it by a definite investing sheath. Moreover, if a subdivision into organs were found only in those brains in which the convolutions were differentiated, then the smooth-brained animals would be excluded from possessing that plurality both of organ and function which would be the heritage of their more fortunate convoluted-brained congeners.   And who can say that the lively and easily-tamed smooth-brained marmoset monkey is one whit more deficient in psychical functions than the baboon or the macaque.

With the convolutions, then, as with other parts, special-
isation of function demands not merely specialisation of ex-
ternal form, but of internal structure.   And it is just in
connection with the existence of special structures in the
different convolutions that we find ourselves so much in need
of fuller and more precise information.   The present state
of our knowledge, indeed, enables us to say that modifications
in the size, in relative numbers, and to some extent also in
the shape of the nerve cells, do undoubtedly occur in various
of the convolutions, and in so far these modifications lend
support to the view that functional differences may exist
between them.   The modifications in relative numbers and
in size are, however, of a nature to support rather differences
in the degree than in the kind of action which takes place
in the convolutions—that they are, in short, quantitative
rather than qualitative.

We have evidence, also, although as yet far from com-
plete, that differences exist in the connections which are
established between the convolutions and the great ganglia
of the base of the brain, and between the different convolu-
tions themselves, through the agency of the internuncial
nerve fibres which form the white core of each gyrus.   In
the differences in their commissural connections we may
look, I think, for an argument in favour of the existence of
functional differences, just as, in the study of the various
members of a group of muscles, we obtain a guide to their
action by examining the attachments of their terminal
tendons.   Similarly, the fact that communications are es-
tablished between certain convolutions, and not between
others, points to the inference that certain gyri are not
only anatomically but physiologically associated directly
with each other ; and it is possible not only that particular
combinations of convolutions, through an interchange of
commissural fibres, may condition a particular state of
intellectual activity, but that these combinations associate
various convolutions together in the performance of a given
intellectual act, just as in the muscular system several muscles
are as a rule associated together for the performance of
a given movement.   It seems to me that the study of the

deeper connections of the convolutions offers a more pro-
mising line of enquiry, in its bearings on the question of the
functional independence of the convolutions or of groups of
convolutions, than an investigation into the arrangement
and physical properties of the nerve cells.

But if the association of separate mental faculties with dis-
tinct convolutions were satisfactorily determined, we should
be no nearer the solution of the mysterious problem, why the
energy evolved from the molecular changes taking place in
one group of nerve cells produces one kind of intellectual
phenomena, and from another group another kind. Or why
from one brain proceeds a wealth of fancy and imagination
which stamps its producer as a true poet ; from another specu-
lations and discoveries on the constitution of mind and matter,
which mark an epoch in science and philosophy ; from a third
the merest commonplaces, or even absurdities, which prove
their author to be a mediocrity or a fool; or why man, more
than the brutes which perish, is endowed with a self-conscious-
ness, with the power of distinguishing good from evil, right
from wrong. All these are problems which lie beyond the
range of anatomico-physiological enquiry, and regarding
them we might indeed say, in terms not unlike those used by
the prophet of old, that eye hath not seen, or ear heard, and
it hath not entered into the mind of man to understand.

EXPERIMENTAL RESEARCHES

IN

CEREBRAL PHYSIOLOGY AND PATHOLOGY.

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THE objects I had in view in undertaking the present re-
search were twofold: first, to put to experimental proof the
views entertained by Dr. Hughlings Jackson on the patho-
logy of Epilepsy, Chorea, and Hemiplegia, by imitating
artificially the 'destroying' and 'discharging lesions' of
disease, which his writings have defined and differentiated;
and, secondly, to follow up the path which the researches of
Fritsch and Hitzig (who have shown the brain to be
susceptible to galvanic stimulation) indicated to me as one
likely to lead to results of great value in the elucidation of
the functions of the cerebral hemispheres, and in the more
exact localisation and diagnosis of cerebral disease.

I have to thank Dr. Crichton Browne for kindly placing
at my disposal the resources of the Pathological Laboratory
of the West Riding Asylum, with a liberal supply of pigeons,
fowls, guinea-pigs, rabbits, cats, and dogs for the purposes
of my research.

Though the present paper is not devoid of results of im-

portant signification in many directions, I would not have it regarded as more than a mere preliminary instalment of a more extended and complete investigation clinically, experimentally, and anatomically. The method I intended to pursue in producing destroying lesions, and which I to some extent carried out, was that recommended by Nothnagel,[1] viz. injection of concentrated solutions of chromic acid into the brain through a small hole in the skull by means of a subcutaneous syringe. This has the merit of destroying the brain substance, and of allowing the exact portions destroyed to be accurately defined by the alterations in colour and consistence of the parts with which the solution comes in contact. Though I have found this method of procedure easily carried out, and have succeeded, by injection into the cerebellum, in producing the usual loss of equilibrium and inability to co-ordinate muscular movements, yet similar injections into the substance of the hemispheres did not enable me, so far as I carried this method, to arrive at any very definite results. Both this and the ordinary methods of experimentation by mechanical destruction or excision of parts of the brain, however well they may be carried out and accurately circumscribed, involve the observation of negative phenomena, which, in a subject like cerebral physiology, is necessarily surrounded by great and often insurmountable difficulties. The injection method requires very great care lest diffusion of the destructive liquid take place into parts other than those whose function it is wished specially to study. On several occasions, in experimenting with guinea-pigs and kittens, has this happened to me, causing such a combination of phenomena that analysis seemed hopeless. It seems probable, however, that this method, in combination with the one I have principally followed, may prove of very effectual service in clearing up many doubtful points.

The progress of my research led me to study almost entirely the effects of electrical irritation of the various parts of the brain. We owe to Fritsch and Hitzig[2] a demon-

[1] 'Centralblatt für die medicinischen Wissenschaften,' No. 45, 1872.
[2] Reichert and Du Bois-Reymond's 'Archiv,' 1870, p. 300, et seq.

stration of the fact that the brain is not, as has been
generally stated, insusceptible to every kind of irritation.
They showed that irritation of the anterior parts of the
brain by a constant current gave rise to certain movements
on the opposite side of the body, and they attempted to
localise in certain definite points the centres for such
muscular movements. Their researches in this direction
were not carried very far, nor do they, I think, clearly
define the nature and signification of the results at which
they arrived. They adduce ample evidence for regarding
the movements that took place as dependent on irritation of
the hemispheres themselves, and they also observed that
irritation proceeded principally, if not exclusively, from the
anode. Induction currents they did not employ to any ex-
tent, and their results by this method did not give them
anything very definite or satisfactory as regards localisation
of function. In my own experiments I have employed
faradisation exclusively, and have found it possible by this
method to produce localised irritation of various parts of the
brain with the utmost exactitude, as well as to induce dif-
fused irritation of the whole of the hemispheres.

For the purposes of stimulation I employed one Stöhrer's
cell (with carbon and zinc elements), and the induced current
of the secondary coil of Du Bois Reymond's magneto-electro-
meter. This allows of tolerably exact graduation of the
strength of the current by sliding the secondary coil along a
measured scale. As a rule, the current was not stronger
than could be borne without great discomfort on the tip of
the tongue. I have in most cases stated the distance of the
secondary from the primary spiral; and it will be seen
that very considerable variations in the strength of the
current are requisite, in order to produce the same effects at
different times in the same animal and in different animals.
The excitability of the brain is exceedingly liable to altera-
tion under various conditions. The shock of the severe
operations requisite for displaying the surface of the brain
to the necessary extent, entailing as they do considerable
hæmorrhage from the sinuses of the diploe and dura mater,
depress the excitability of the brain in a very remarkable

degree. After great hæmorrhage, and when the brain ceases to pulsate, even long before the death of the animal, the strongest currents fail to stimulate, or, if they do, the phenomena are so complicated with the effects of conducted currents that no satisfactory inferences can be drawn as regards localisation. For this reason it is well to expose the brain in detail, determining the functions in each successive part exposed, rather than to lay bare a whole hemisphere at once. As the operations necessary to reach the anterior and under surface of the hemispheres involve extirpation of the eyeball, removal of the roof of the orbit, and of the zygoma, with reflection of the temporal muscle, the shock and hæmorrhage have so far depressed the excitability of the brain that the experiments fail. This has happened to me several times when I had in one operation laid bare the whole of one hemisphere. The excitability is also greatly lowered by chloroform and ether, under the influence of which the animals have to be experimented on, not only from humane motives, but to exclude the complication of voluntary or reflex movements.

In all cases it is absolutely necessary to remove the dura mater and avoid irritating it during the process of experimentation. The dura mater is so excessively sensitive that pinching it, or stimulating it by the electric current, is sufficient to cause violent movements.

The stimulation of the surface of the hemispheres by electricity causes functional hyperæmia in the parts irritated. The mere contact of the electrodes with the surface causes increased vascularity, and often induces profuse hæmorrhage from the sinuses which, in the quiescent state of the brain, had ceased to bleed. This effect is very marked in the comparatively soft cortical substance of the brain in rabbits. Several applications of the electrodes to the exposed part of the brain in these animals converts it rapidly into a kind of fungus hæmatodes. This is still more pronounced in the case of pigeons. Electric stimulation causes in them such a general oozing, that the parts are soon rendered obscure and indistinguishable. Nothwithstanding this very marked hyperæmia, the hemispheres and cerebellum of pigeons and

fowls, at least the two or three I have examined, remain ab-
solutely insensible, so far as outward effects are concerned,
to the electric stimulation. I have repeated the experiments
several times, using the full strength of the coil, and have
failed to cause stimulation, either of the surface of the hemi-
spheres or of their deeper parts. This, taken with the fact
discovered by Weir Mitchell that pigeons and fowls are insus-
ceptible to the action of opium, would seem to point to some
peculiarity of the nervous centres of these animals, and pos-
sibly that the same cause underlies the two sets of phenomena.
For this reason electric stimulation does not seem suited for
determining the function of the cerebrum or cerebellum in
birds.

In order to carry out the operations necessary for exposing
the brain and observing the effects of stimulation, I have
simply narcotised the animal, and extended it on a board with
the abdomen downwards, and then during the stimulation
relaxed the cords so as to give the head and legs free play.
The brain was exposed by trephining, and the surface sub-
sequently extended by the use of the bone forceps. The
hæmorrhage from the sinuses I have found most effectually
controlled by cotton wool, which can be insinuated into the
various bleeding orifices. The electrodes used for irritation
were simple copper wires doubled at the end, and slightly
rounded so as to avoid laceration of the parts to which they
were applied. They were insulated up to the point. One or
two preliminary experiments were made, simply with a view
to observe the effects, if any, of the vital irritation of the sur-
face of the hemispheres consequent on free exposure, by re-
moval of the greater part of the bone and the subjacent dura
mater. The following were the results observed in the case
of a guinea pig whose left hemisphere was exposed to the ex-
tent indicated in the woodcut (fig. 1), accurately marked by
painting it with chromic acid before removal from the skull.

EXPERIMENT I.—Medium-sized guinea-pig. Narcotised with chloroform,
and the greater part of left hemisphere exposed. The animal was then
placed on the floor and watched. On recovery from stupor the body be-
comes curved from left to right, the head touching the tail. Active move-
ments of the forelegs are made, causing the animal to rotate round from left
to right. Sometimes the animal makes violent struggles and falls on its

back. When the muscles of the right side are forcibly overcome, the state of pleurosthotonus returns on removal of the resistance. When placed on its right side the animal makes rapid movemeuts with both fore and hind legs, as in running, but is unable to alter its position. When placed on its left side it becomes curled from left to right as before, and regains its feet. The pleurosthotonus and movements of the legs occasionally remit. Half-an-hour after the operation the animal is able to rest quietly on its feet, but with the head directed towards the tail from left to right. The animal was then, 45 minutes after the operation, again narcotised, and submitted to electric stimulation, but death took place, probably from an overdose of chloroform, during the first application of the electrodes. Apparently the brain retained its excitability, but I discovered that the movements induced by stimulating different parts, and which were on the left side, were in reality due to conducted currents from too strong stimulation.
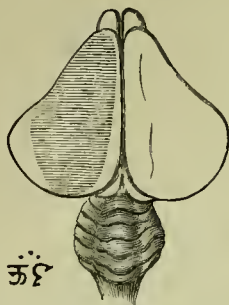
Fig. 1.—Upper surface of the brain of guinea-pig. The part shaded indicates the extent to which the left hemisphere was exposed.

The inferences I subsequently drew from the experiment were, that the vital irritation, consequent on exposure of the hemisphere, acted on the muscles of the opposite side of the body through the corpus striatum, causing tetanic spasm and pleurosthotonus. (See below, p. 62.)

The next experiments were undertaken with a view to ascertain whether long continued faradisation of the cerebral hemispheres would induce such a condition as to determine convulsions of an epileptiform character, similar to the discharging lesions caused by tumours pressing on the surface of the brain; and, if so, to ascertain the usual march of the spasms.

Several rabbits and cats were employed for this purpose, and the results were similar in all. In the subsequent experiments for the purposes of more exact localisation of function, it will be seen that epileptiform convulsions could be induced with the greatest readiness in all the animals experimented on.

It may be mentioned here, once for all, that before and throughout all the following experiments, ether or chloroform was administered.

EXPERIMENT II.—Full-grown rabbit. The left hemisphere was exposed by the removal of a triangular portion of the skull corresponding to the parietal region of the hemisphere, the apex of the triangle not extending beyond the posterior margin of the orbit. The electrodes were applied to the anterior and posterior portions of the exposed surface.

Obs. 1.—Secondary at 8 cm. Electrodes applied for five seconds.

During irritation the animal remained perfectly still to all appearance. After the lapse of fifteen seconds twitchings began in the muscles of the right side of face, the head gradually turned to the right, until the nose pointed directly upwards over the right shoulder, and the jaws became affected with violent clonic spasms. This fit lasted several seconds, but the time and exact march of the spasms was not more accurately noticed in this instance.

After the animal had been allowed to recover and rest for a few minutes the electrodes were applied as before for five seconds.

Obs. 2.—During the irritation, and for twenty-one seconds after removal of the electrodes, the animal remained still. Then the right lip began to twitch. The twitchings gradually invaded the whole right side of the face, the head became convulsively drawn to the right until the nose pointed over the right shoulder, the jaws worked, and the animal continued to be affected with clonic spasms, in the right side of face and neck for 1 minute 32 seconds, after which the fit entirely ceased.

Obs. 3.—Irritation as before, and effects essentially same as in Obs. 2.

Obs. 4.—The secondary was now pushed up to 6 cm., and the electrodes applied for five seconds.

After forty seconds the right lip began to twitch. In fifty-five seconds twitching in the right fore-paw; in one minute ten seconds, right lip twitching violently and jaws working; in one minute thirty seconds, head drawn over right shoulder; in one minute forty-six seconds the fit stopped suddenly, and animal appeared to wake up from a state of stupor.

Several other observations were made, but as the results were in all respects similar to those already given, they need not be further detailed.

After the animal had been untied and placed on the floor, it was seized spontaneously with the most violent fit that had occurred, lasting for two minutes, and affecting the whole of the right side of the body, face, neck, and legs.

A slight fit, affecting only the right side of the face and neck, again occurred half-an-hour afterwards, but only of transitory duration, and the animal recovered perfectly. Its subsequent history will be alluded to further on. Another rabbit similarly operated on yielded results of a similar nature.

EXPERIMENT III.—The next experiment was made on a large, strong cat. The skull was removed and the left hemi-

sphere exposed to the extent shaded in fig. 2, as was ascertained by painting the exposed surface with chromic acid previous to entire exposure of the brain after death. A sketch was made of the convolutions exposed, and the points in which the electrodes were applied accurately marked. The animal was only partially narcotised.
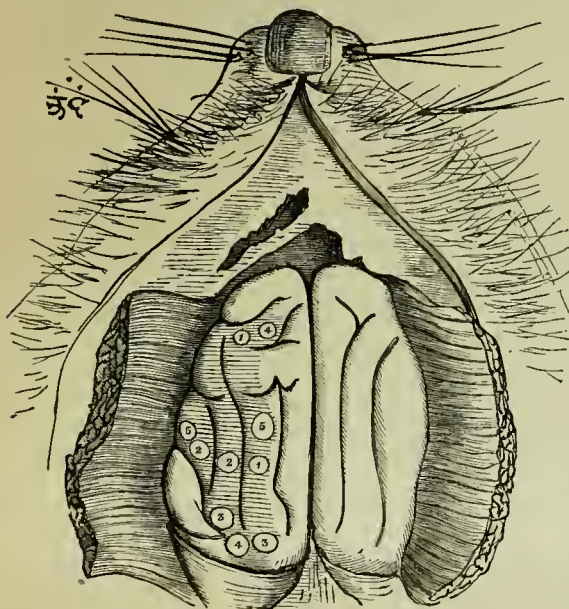
Fig. 2.—View of the brain of a cat *in situ.* The shaded part indicates the extent to which the left hemisphere was exposed. The numerals (1–1), (2–2), (3–3), (4–4), (5–5) indicate the different positions of the two electrodes as described in the text.

Obs. 1.—Secondary at 5 cm. Electrodes applied on the points marked 1–1 for five seconds. A fit occurred instantaneously after the removal of the electrodes, and lasted thirty seconds. The spasms, which were of a clonic character, began in the right eyelid and right lip, and gradually affected the whole of the right side of the face. The head next became drawn to the right, with the nose pointing to the right shoulder. The right fore-leg and right shoulder were next convulsed, and afterwards the right hind-leg, the tail at the same time becoming erect and convulsed. The convulsions passed off in the order in which they had come on.

Obs. 2.—Ten minutes after the first observation the electrodes were next applied for five seconds to the points marked 2–2 (fig. 2).

Almost before the electrodes were withdrawn the head was spasmodically drawn to the right. Then followed spasmodic twitching of the right eyelid and right ear, the head remained twisted to the right by clonic spasms.

There were no movements in the legs. The pupils were observed to be widely dilated. The whole fit lasted thirty-seven seconds.

Obs. 3.—After other ten minutes had elapsed, the electrodes were applied for five seconds to the points marked 3-3 (fig. 2).

The head was immediately turned to the right, but it was doubtful whether the movement was spasmodic or voluntary, as the animal was only partially insensible.

Twitching next began in the right lower eyelid and right lip, followed by twitching in the right ear, and slight twitching in the eyelid and lip of the opposite side.

After this fit had passed off the animal lay in a stupid, half-sleeping condition on its left side. During this time there was some degree of rigidity and quivering of the right fore and hind legs.

Obs. 4.—Ten minutes having again been allowed to elapse, the electrodes were applied for five seconds, as before, to the points marked 4-4 (fig. 2).

During the application of the electrodes, the animal shrunk together, and immediately after removal of the electrodes, a severe unilateral fit, affecting the right side, came on, and lasted for one minute.

During this fit there were violent clonic spasms of the face, the neck, the jaws, the legs. The tail became erect and violently twitched from side to side. The jaws were spasmodically opened and closed, the tongue protruded and frequently clenched between the jaws, while copious frothy saliva exuded from the mouth. The face and jaws first ceased being convulsed, then the clonic spasms of the lip gradually abated.

Obs. 5.—Seven minutes after the last observation the electrodes were applied for five seconds to the points marked 1-1, as in the first observation, but the posterior electrode was placed a little further back. Immediately on removal of the electrodes, clonic convulsive spasms occurred in the right eyelid, lip, and right side of the face, and spread next to the right shoulder and fore-leg, which were violently convulsed. The spasms next attacked the right hind-leg and tail, the tail becoming erect and convulsed nineteen seconds from the commencement of the fit. During the fit, the head was jerked backwards and to the right, the jaws worked furiously, the tongue was frequently protruded, and copious frothy saliva flowed from the mouth. The pupils were widely dilated. The whole fit lasted one minute fifty-nine seconds, the animal waking up from an apparent state of unconsciousness.

Obs. 6.—Eight minutes having elapsed, the animal being in a quiescent state, the electrodes were again applied for five seconds to the points marked 5-5 (fig. 2). Before withdrawal of the electrodes twitching began in the right eyelid, and the head was drawn to the right. Twitchings then occurred in the right lip and right ear, and then in the muscles of the right side of the neck. A few twitchings were observed in the right legs, occurring in the hind-leg last. There were no movements of the jaws or tail. The fit lasted one minute five seconds.

Several other observations were made with similar results, the most severe fits being induced when the electrodes were applied at the anterior and posterior extremities of the exposed surface.

Without detailing further experiments as to the artificial production of epilepsy, as the occurrence of such fits will be alluded to in the course of subsequent experiments related below, or attempting to account for their nature, I would only call attention to the fact that in all cases, whether the fits were partial or more general, the immediate antecedent was an excited hyperæmic condition of the cortical matter of the hemispheres. The irritation was entirely confined to the surface of the hemispheres, the electrodes being simply applied without causing mechanical or deep seated lesion in any case. And the peculiarity observable in the case of the rabbit in Experiment II. was that it took a distinct interval of time after the withdrawal of the stimulation before the condition of the grey matter had reached the pitch of tension requisite for an explosive clonic discharge. This of itself is sufficient to show that the effects were not due to con-ducted currents or direct stimulation of the motor nerves of the muscles, but to an abnormal excitability or irritability of parts whose function, it might be inferred, was to initiate those changes which would result in normal contraction of the muscles affected. Certain variations in the order of the phenomena, according to the position of the electrodes on the hemispheres, led me next to endeavour to ascertain whether it might be possible accurately to define and localise the points from which individual muscles, or groups of muscles, might be set in action without inducing general convulsions. And, though I was aware that Fritsch and Hitzig had succeeded in producing definite muscular con-tractions from galvanisation of certain regions in the cere-bral hemispheres, I was surprised to find with what degree of definiteness the approximation of the electrodes on certain particular spots called forth muscular movements, on the opposite side of the body, of a determinate character. Certain preliminary results having been arrived at in the partially exposed hemisphere of a cat, more elaborate experi-ments were next undertaken to expose the whole hemisphere, and try the convolutions in detail.

Owing to the severity of the operation, and the great de-pression of the excitability of the brain from hæmorrhage,

several failures were experienced, and in only one was I able
to carry the exploration over the whole hemisphere. Partial
confirmatory observations, however, were made on three or
four others. (See also p. 47.) As already mentioned, the
surface of the brain must be exposed in detail if one is not
to run the risk of losing everything. The experiments are
recorded, therefore, in the order in which they were made,
and without reference to any principles of classification or
grouping. For the purpose of accurately noting the points
on which the electrodes were applied, the hardened brain of
a cat was placed in front of the operator; the parts exposed
were carefully noted and compared, and the points stimu-
lated marked correspondingly on the hardened brain. In
this way an exact record was kept of what was done. The
results of stimulation of the individual convolutions were
only noted after the effect had been produced several times
in succession without variation, and in the presence of my
undermentioned medical friends.

Without committing myself to any very definite state-
ments regarding the homology of the convolutions, I have in
addition to referring to the numbers in the figures (fig. 3,
&c.), used for convenience in description a nomenclature
founded on that of Leuret and Gratiolet.

On account of the more apparent continuity, both in the
cat and dog, of the three upper external convolutions from
the frontal region backwards, and downwards to the temporo-
sphenoidal region, I have preferred to call them the superior,
middle, and inferior external convolutions, and to term their
anterior divisions in advance of the fissure of Sylvius the
frontal convolutions, or frontal divisions of the superior,
middle, and inferior external. The peculiar gyration round
the crucial sulcus, A (figs. 3, 4, &c.), in the superior external,
which is much more highly developed in the dog than in the
cat, I have called the *sigmoid* gyrus, borrowing a term used
by Mr. Flower.[1] The convolution which arches over the
fissure of Sylvius, I have called the supra-Sylvian (based on
the nomenclature of Owen), instead of calling it an external

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[1] ' Proceedings of the Zoological Society of London, November, 1869.' Anatomy
of the Proteles.

convolution. The orbital region of the brain I have called, with Leuret, the supra-orbital convolutions, but have also used terms to signify their apparent continuity with the various frontal convolutions.

I have designated the parts of the convolutions occasionally by names indicating their position with respect to the cranium, using a nomenclature borrowed from the writings of Gratiolet, Huxley, Turner, &c.

The inner convolution I have, as usual, called the marginal or calloso-marginal, and its continuation downwards and backwards the hippocampal convolution.

A reference to the figures (various) will indicate the position of the sulci and the convolutions, with their respective names as used in the description.

Experiment IV.—The greater part of the right hemisphere of a full-grown, strong cat is exposed. The animal lies breathing quietly in the semi-narcotised condition. Secondary spiral at 8 cm., one Stöhrer's cell.

Obs. 1.—Electrodes applied to point (1), fig. 3, on the sigmoid gyration of the frontal division of the superior external convolution, just anterior to the crucial sulcus.

There is slow distinct flexion of the phalanges of the left fore-paw, and elevation of the left shoulder. Several times repeated.

Obs. 2.—Electrodes placed on point (2), figs. 3, 4, just posterior to the crucial sulcus.

Retraction and adduction of the left fore-paw.[1] At the same time, as ascertained subsequently, the left leg is advanced from its extended position.

Obs. 3.—Electrodes on point (3), figs. 3, 4, *i.e.* the posterior part of the frontal division of the superior exteral convolution.

No result was observed in this case either with secondary coil at 8 cm. or pushed up to 5 cm.

Obs. 4.—Electrodes applied to points marked (1) and (3), figs. 3 and 4.

During the application there is combination of the previously-described movements, viz. flexion of the toes of the left paw, with retraction and adduction of the limb, the left hind-leg being advanced.

Obs. 5.—Electrodes applied to point (4), figs. 3 and 4, about the middle of the superior external convolution.

Immediate corrugation of left eyebrow, and drawing downwards and inwards of the left ear.

[1] Such, apparently, is the movement when the animal remains tied down; but when the animal is untied, and the leg allowed free play, stimulation of this point causes the shoulder to be raised, and the limb adducted, exactly as when a cat strikes a ball with its paw.

Obs. 6.—Electrodes applied to point (5), figs. 3 and 4, *i.e.* the posterior angle of the superior external convolution.
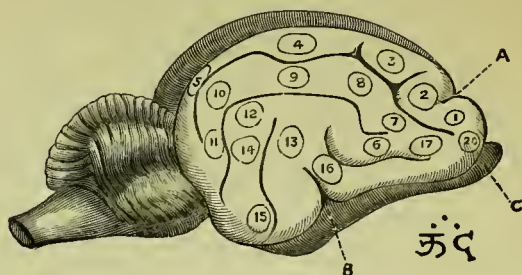
Fig. 3.—View of the right hemisphere of brain of cat. A indicates the crucial sulcus, B the fissure of Sylvius, C the olfactory bulb and tract. The circlets and included numbers indicate the position of the electrodes as described in the text. The numbers (1), (2), (3), (4), (5) are on the superior external convolution ; (1), (2) and (3) on its frontal division. The numbers (7), (8), (9), (10), (11) are on the middle external convolution ; (7) and (8) being on the frontal division. The numbers (6), (12), (14), (15) are on the inferior external. The number (13) is on the annectent gyrus between this and the supra-Sylvian convolution, of which (16) indicates the anterior part.

The animal exhibits signs of pain, screams and kicks with both hind-legs, especially the left. This result was observed several times in succession.

Obs. 7.—Electrodes applied to point (6), figs. 3 and 4, at the point where the middle and inferior external arise from the external supra-orbital.
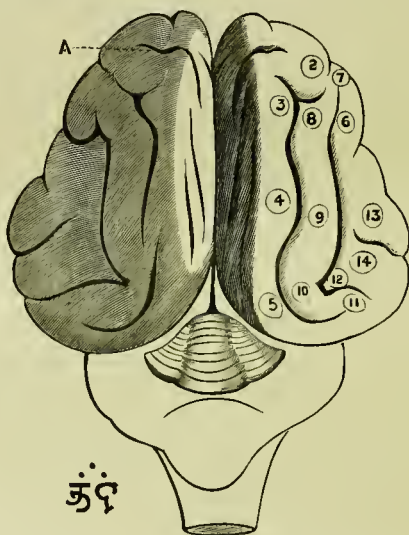
Fig. 4.—Upper view of the same brain.
The letters and numerals indicate the same as in the preceding figure.

Clutching movement of the left paw, with protrusion of the claws. The movement in this case was perfectly distinct from that described in Obs. 1. The two movements were repeated and contrasted several times in succession.

Obs. 8.—Electrodes on point (7), figs. 3 and 4, the anterior frontal extremity of the middle external convolution.

Immediate elevation of the left upper lip, and closure of the left eye.

Obs. 9.—Electrodes on point (8), figs. 3 and 4, on the middle external convolution, post-frontal part.

Immediate retraction of the left ear, drawing up of the left nostril and cheek, with forcible closure of the left eye.[1]

Obs. 9'.—Electrodes on point (9), figs 3 and 4 ; the middle of the middle external convolution (parietal region).

Immediate drawing of the head to the left side.

Obs. 10.—Electrodes on point (10), figs 3 and 4, on the postero-parietal region of the middle frontal convolution.

Rotation of head to the left as before

Obs. 11.—Electrodes at point (11), further back to same convolution, *causes same effect, viz., rotation of head to the left.*

Obs. 12.—The electrodes were again applied to the point (5), figs 3 and 4.

The animal exhibited signs of pain, screamed and kicked out with its left hind-leg, at the same time turning its head round and looking behind in an astonished manner.

*Obs.* 13.—Electrodes on point (12), figs. 3 and 4, the recurved portion of the inferior and external convolution.

Rotation of head to the left.

Obs. 14. Electrodes on point (13), figs. 3 and 4, on the annectent gyrus between the inferior frontal and supra-Sylvian convolution.

Twitching backwards of left ear, and rotation of the head to the left and slightly upwards.

Obs. 15.—Electrodes on point (14), figs. 3 and 4, the descending limb of the inferior external convolution.

Head drawn to the left without elevation, along with some movement of the ear, and upper eyelid (?).

Obs. 16. — Electrodes again applied to the point (5) as in Obs. 6 and 12, with precisely similar results.

Obs. 17.—Electrodes again applied to the point (1), figs. 3 and 4, with same result as given in Obs. 1.

Fig. 5.—Orbital surface of the same brain. The letters have the same signification as in fig. 3, as well as some of the numerals. No. (19) is on the orbital extremity of the superior external; (17) is on the orbital extremity of the conjoint middle and lower external ; while (18) is on the anterior or orbital extremity of the supra-Sylvian convolution. D is on the hippocampal expansion of the olfactory tract.

Obs. 18.—Electrodes to point (15), fig. 3, the secondary coil now moved

[1] Stimulation of this point, as well as of the next two, causes movements of the eyeballs. Usually they were directed to the opposite side, but some variations occurred, and further definition has not yet been arrived at. These results are noted here, but they were only ascertained in subsequent experimentation on two other cats, and therefore are not mentioned in the experiments related below.

up to 4 or 8, failed to produce effect. No. 15 is on the external surface of the temporo-sphenoidal lohe, behind tbe fissure of Sylvius. *Repeated closing and opening of the jaws.* Movements of the jaws were constantly evoked by irritation of tbe regions in this neighbourhood. Along with the movements of tbe jaws, the tongue was seen often to be protruded and retracted, but exact definition of the points for the various movements was not arrived at at the time (Vide p. 47).

Obs. 19.—Electrodes on point (16), fig. 3, corresponding to the anterior limb of the supra-Sylvian convolution, and is just anterior to the fissure of Sylvius.

Rotation of the head to the left, closure of the left eye, and drawing down-wards of the left corner of the mouth.

Wbile tbe temporo-sphenoidal gyri were being further exposed, the animal bit angrily, and gnawed its own legs. It did the same generally after irritation of the same parts.

Obs. 20.—Electrodes on point (17), figs. 3 and 5, with secondary coil at 4, 5, and 6 cm. respectively. Point 17 is on tbe external supra-orbital, or it may be called the frontal extremity of the third or inferior external convo-lution.

In every case restlessness, opening of the mouth, and long-continued cries as if of rage or pain.

Obs. 21.—Electrodes (secondary 6 cm.) on tbe point marked (18), fig. 5. This point is on the internal supra-orbital, or rather tbe frontal extremity of the supra-Sylvian gyrus.

The animal suddenly starts up, throws back its head, opens its eyes widely, lashes its tail, pants, screams and spits as if in furious rage.

This ohservation was several times repeated. The animal at the end of each such manifestation sank down into its stupid condition.

Obs. 22.—Electrodes on point (19), fig. 5, the lowest point of the superior external as it lies on the orbit.

Immediate jerking of the head backwards as, if from irritation applied to the nostrils. Again, with stronger irritation, tbe head is thrown back-wards, the left hind leg advanced, and the tail lashed from side to side.

Obs. 23.—Electrodes on point (20), figs. 3 and 5, the frontal tip of the superior external convolution.

Sudden contraction of the muscles of the front of the chest, neck, and depres-sors of lower jaw. The animal pants in a very distinct manner. Similar results followed several applications of the electrode.

Obs. 24.—The electrodes were now slipped within the longitudinal fissure, and applied to the marginal convolution at several points. No result could be observed.

The animal was now becoming exhausted, and therefore it was considered possible that tbe excitability of the brain was too depressed to react to stimulation. The other parts, however, at the anterior extremity still remained excitable.

In order to determine this point with greater certainty, I made on a subsequent occasion an experiment on another cat, laying bare especially the marginal convolution in its full extent.

Application of tbo electrodes along the whole of tbe inner surface of the

longitudinal sulcus posterior to the crucial sulcus (Sillon crucial ; Leuret and Gratiolet) and downwards and backwards along the hippocampal gyrus, failed to produce any effect. When the irritation was made stronger, and continued for some time, an epileptiform seizure was induced, the head being thrown back spasmodically. The only effect observed, on irritating the marginal anterior to the crucial sulcus, was the turning of the head to the opposite side. The other convolutions at the anterior aspect gave results identical with those described above, in so far as the experiments were carried.

Obs. 25.—Application of the electrodes by pushing them under and behind the temporo-sphenoidal gyri, gave no results. By touching rapidly in succession the various points already defined, the animal was made to execute a series of choreic movements, viz., spasmodic twitching of all the muscles and groups of muscles of which these were the centres, the twitching often lasting for several seconds. The electrodes were lastly applied to the anterior and posterior extremities of the exposed brain, with the effect of producing unilateral left-sided epileptic convulsions so long as the stimulation was kept up. The excitability of the brain was now well-nigh exhausted, and it entirely disappeared four hours after the commencement of the experiment, during which period the exploration was kept up uninterruptedly.

Before analysing or attempting to generalise from these data, I next proceed to record the results of an experiment on another cat, made with a view to ascertain whether the two sides of the brain were symmetrical. This time the left hemisphere was exposed, and the position of the electrodes on the various convolutions made as nearly as possible on the points corresponding to those already determined on the right hemisphere. The results were noted down at the time, and as those of the previous experiment had not been analysed, there was no bias to interfere with accurate representation of facts. Though in this case I was not so successful in maintaining the life of the animal and the excitability of the brain so long as in the former case, and therefore could not explore every point, yet the remarkable uniformity of the results arrived at, as will be seen by comparison of the two experiments, give every reason to suppose that complete symmetry exists.

EXPERIMENT V.—Cat. Left hemisphere partially exposed.

Obs. 1.—Secondary at 6 cm. Electrodes on point corresponding to (8), fig. 3. Immediate closure of the right eye. A comparison of this result with Obs. 9, Experiment IV., indicates, though to a less extent, the same muscular movements.

Obs. 2.—Electrodes on points immediately above (13) and (14), figs. 3 and 4.

Head immediately turned to the right.

Obs. 3.—Electrodes on point corresponding to (13), figs. 3 and 4.

Retraction of right ear, and rotation of the head to the right.

A comparison of this with Obs. 14, Experiment IV., will show the complete uniformity and symmetry.

Obs. 4.—Electrodes on point corresponding to (5), fig. 3. Just as occurred in Obs. 6, Experiment IV. The animal exhibited signs of pain, and looked round and backwards to the right.

Obs. 5.—Electrodes on point corresponding to (2), figs. 3, 4.

Immediate retraction (with adduction) of right paw. The effect was exactly such as described in Obs. 2, Experiment IV.

Obs. 6.—Electrodes on point corresponding to (7), figs. 3 and 4.

Immediate elevation of the right lip and whiskers. Here also the movement was exactly similar to that recorded in Obs. 8, Exp. IV., but the extent was not so great, and therefore the eye was not closed.

Obs. 7.—The excitability of the brain being considerably depressed, the secondary coil was pushed up to 5 cm.

Electrodes on point corresponding to (6), figs. 3 and 4.

Clutching movement of right paw.

The electrodes were then pushed a little lower down in front, but the exact situation was not defined.

The animal immediately opened its mouth.

This result would agree with what was obtained by placing the electrodes on the point (17), figs. 3 and 5.

The experimentation, however, came to an end by the exhaustion and death of the animal.

The exact duration of the whole experiment was not noted.

The remarkable if not absolute uniformity of the results obtained by stimulating the corresponding convolutions on the right and left side respectively, even though the comparison unfortunately could not be carried out to a complete extent, is quite sufficient to justify the inference that the two sides of the brain are symmetrical in their function.

As nearly as possible, the whole hemisphere was exposed, and the electrodes applied in close proximity, usually about a quarter of an inch or less apart from each other, on the various convolutions, while regard was paid not to complicate the results by applying them in too close approximation to the centres already determined. Further experimentation may enable us to localise with a still greater degree of precision the exact extent of the convolutions which preside over the various movements.

The general result of the muscular action only has been noted, without any special attempt having been made to differentiate the various muscles called into action.

Some phenomena which appeared to me so curious when writing the above, led me to repeat the experiment on another cat, the chief purpose being to define more exactly the results of stimulation of the orbital region, and the region of the fissure of Sylvius.

EXPERIMENT V'.—The left hemisphere was exposed, and the electrodes applied in succession to the points corresponding to those already described.

Stimulation of the middle external convolution just posterior to the sulcus marking off the frontal region, and nearly corresponding to point (8), fig. 3, caused elevation or retraction of the right ear and drawing up of the lower eyelid so as to close the eye. Slightly in advance of this point, stimulation caused drawing up of the right cheek, forcible closure of the right eye, and rather a drawing forward and downward of the right ear.

Here a more exact differentiation of the conjoint movements of the eyelids and ears will be observed, but the results are quite in harmony with the former observations, both of Experiments IV. and V.

Stimulation of the sigmoid gyrus posterior to the crucial sulcus caused elevation of the right shoulder with retraction and slight adduction of the right fore paw.

Here, again, the previous results in both cases were confirmed.

After this stimulation, the shoulder and limb continued for a minute or two to twitch in a choreic-like manner, the same movements being carried out, but in spasmodic succession.

Stimulation of the parietal regions, as indicated in the figures, caused rotation of the head to the right. Stimulation of the recurved portions of the external convolutions failed to produce movements, but generally the animal uttered cries. I am inclined to regard these as the direct result of the stimulation, and not as merely accidental concomitants.

The orbital regions and the fissure of Sylvius having been exposed, the electrodes were applied to the regions corresponding with (17) and (18), being the anterior extremities of the lower frontal and supra-sylvian convolutions.

In every case, and the experiments were repeated frequently, the mouth was opened in a clonic convulsive manner, and the tongue was protruded, apparently in a straight line, while the animal uttered cries as if in anger or pain, and lashed its tail. The opening of the mouth and movements of the tongue (alternate protrusion and retraction) were always very distinct, so that all doubt as to regions in which these movements are centralised seemed removed. Long-continued stimulation of the region caused the mouth to be opened to its fullest extent in clonic spasms, while the tongue was alternately protruded and retracted.

Chloroform was administered till the animal was in a state of profound stupor. On again stimulating the same region, the opening of the mouth

and movements of the tongue were repeated, but no cries were elicited. Stimulation of the region posterior to the fissure of Sylvius caused the jaws always to be firmly closed.   Opening of the mouth and closure was alternately induced by stimulating tbe former region and this respectively.

One electrode being placed on the anterior extremity of the superior external, and the other on the orbital extremity of the lower frontal and supra-Sylvian, caused the head to be drawn back, and the jaws opened to their fullest extent in a convulsive manner, with movements of the tongue as before.   Here, again, in a striking manner, the observations made in Experiment IV. are further corroborated, while at the same time they are rendered more definite.

I next proceed to relate the results of similar experiments on dogs.   Several dogs died under the influence of chloroform before the experiments could be carried out, and I only succeeded in getting a tolerably complete exploration in one dog and a partial exploration in two others.

The results were, however, so definite, and repeated with such exactitude, that I have no hesitation in regarding them as satisfactory and representative.

EXPERIMENT VI.—The left hemisphere of a lively mongrel cur was to a great extent exposed before beginning the faradisation, and the other parts exposed in detail after the function of the previously-exposed portions had been determined.
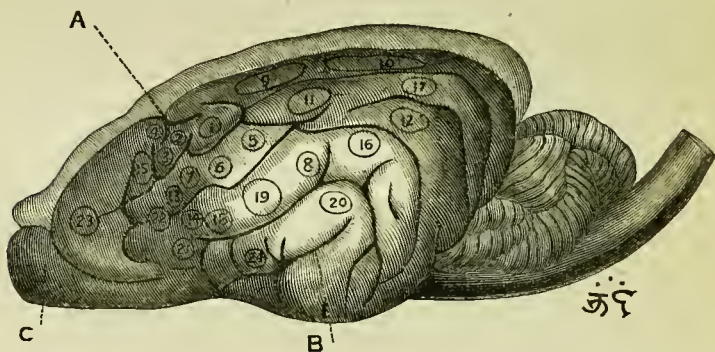
Fig. 6.—Left hemisphere of brain of dog. The letters indicate the same as in the preceding figures.   The numerals (4), (3), (2), (1), (9), (10) are on the superior external convolution ; the first four being on the sigmoid gyrus of the frontal division.   The numerals (7), (6), (5), (11), (12) and (17) are on the middle external ; (17) being on the superior, and (12) on the inferior division of its posterior extremity ; while (7), (6) and (5) are on the frontal division. The numerals (13), (14), (18), (19), (8), (16) are on the inferior external convolution, while (25) and (20) are on the supra-Sylvian convolution.   The other numerals indicate the orbital extremities of the different convolutions, and are alluded to, and their signification given, in the text.

The secondary coil was fixed at 8 cm.

Obs. 1.—Electrodes on point (1), figs. 6 and 7, the posterior part of the sigmoid gyrus of tho frontal division of the superior external convolution.

Adduction of the right fore paw. This action was slight.

Obs. 2.—Electrodes on same gyrus, a little in advance.

Elevation of the right shoulder, adduction of the right paw, with extension of the toes. The right hind-leg is also flexed and advanced.[1]

After the cessation of the stimulation (several times repeated) a unilateral epileptic seizure occurred, in which there was spasmodic twitching of the right eyelid, raising of the right shoulder, and spasmodic elevation of the tail.

Obs. 3.—Electrodes on points (2), (3), figs. 6 and 7, still on the sigmoid gyrus, but just at the extremity of the horizontal division of the crucial sulcus.

In this case the only movement observable was *rotation of the head to the right shoulder.* I have not had another opportunity of verifying this result. It, however, seems to agree with the results of Fritsch and Hitzig's experiments.[2]

Obs. 4.—Electrodes on (4), figs. 6 and 7, the anterior limb of the sigmoid gyrus of the superior external convolution.

Immediate elevation of the right eyebrow and eyelid.

The secondary was now moved up to 7, as the current seemed weaker than before, from some fault in the battery. As a rule, the electrodes could be borne on the tip of the tongue without great discomfort.

Obs. 5.—Electrodes on point (5), figs. 6 and 7, the posterior extremity of the frontal division of the middle external convolution.

The right eye is forcibly closed and the head twitched to the right.[3] Continuous stimulation caused rapid winking of the right eyelids.

Obs. 6.—Electrodes on point (6), figs. 6 and 7, near the middle of the same convolution.

The same result as in Obs. 5, viz., winking of the right eyelid.

Obs. 7.—Electrodes on point (7), figs. 6 and 7, a point on the same mid-frontal convolution just anterior to the last.

The result is the same as in Obs. 5 and 6, but not so well marked.

Obs. 8.—Electrodes on point (8), figs. 6 and 7, the posterior extremity of the frontal division of the inferior external.

The right eye is spasmodically closed.

Obs. 9.—Electrodes on point (9), figs. 6 and 7, on the superior external, behind the sigmoid gyrus.

The tail is moved from side to side, and ultimately becomes rigidly erect. Some contraction of the muscles on the outer side of the right thigh was also observed. Movements of the tail were induced repeatedly by application to this point, and for some extent behind it.

After this experiment another epileptic attack occurred, characterised by

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[1] On a subsequent occasion these movements were more differentiated. The leg centre alone was found to be a little higher up nearer the median fissure, and in the gyrus connecting this one with the superior external. It is not specially marked in the cut by a numeral. It is referred to as (†).

[2] In another later experiment I found that stimulation of this point, as also of (3), caused the opposite eye to turn inwards, and the pupil to dilate. Very slight similar action was observable in the eye of same side.

[3] In a later experiment it was found that the eyeballs were also both directed outwards and slightly downwards, and the pupil distinctly contracted.

clonic spasms of the right eyelids and shoulder, with rigid erection of the tail.   The whole fit lasted twenty seconds.

The fit seemed limited to the muscular movements, whose centres had already for some time been under repeated stimulation.

Obs. 10.—Electrodes to points included in circle (10), figs. 6 and 7 on the middle of the superior external convolution posterior to the tail centre.

Several applications elicited only cries as if in pain.   The secondary coil was moved gradually up to 3 cm., under the idea, which seemed to be borne out by other experiments, that in order to produce movements, if any were represented in this part, stronger stimulation was necessary than in the more anteriorly placed centres.   The results, however, did not vary.
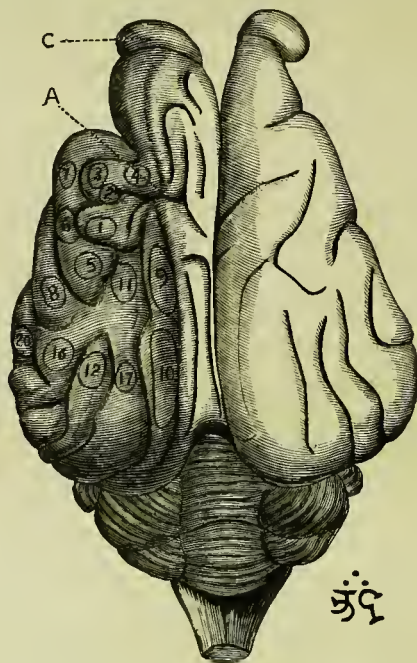
Fig. 7.—Upper view of the same brain.   The letters and numerals have the same signification as in fig. 6.   The calloso marginal gyrus is exposed on the left hemisphere by drawing asunder of the two hemispheres.

As the animal was emitting cries also during the intervals of stimulation, it was thought that the cries of pain might not be the result of the stimulation, but their immediate intensification on the application of the electrodes seemed to be the direct result of irritation.   On this point, however, I should not like to pronounce too dogmatically, though the results obtained in the cat by stimulation of the homologous parts would seem to indicate this to be a sensory region.

Obs. 11.—Electrodes on (11), figs. 6 and 7, about the middle of the middle external convolution, and just behind the point (5).

Spasmodic contraction of right eyelids.

After this the animal had another unilateral convulsion, affecting the whole of the right side. The face, neck, legs and tail, were violently convulsed.

The same point was again stimulated with a stronger current, and again an epileptic seizure occurred. It began with twitching of the right lower eyelid, spread to the shoulder, thigh and tail, which became erect, and spasmodically twitched from side to side. The twitching ultimately attacked the left lower eyelid. The animal during the fit was apparently completely unconscious.

The animal after this became very restless. It was again rendered quiet by another administration of ether. During the deep narcosis the parts (5) (6) and (7) were again stimulated without any result. This was due without doubt to the depression of the excitability of the brain caused by the anæsthetic, and by former applications.

Obs. 12.—Secondary at 4 cm.

Electrodes applied on point 12, figs. 6 and 7, the recurved portion of the lower division of the middle external convolution.

The head was moved to the right, but as the animal was still restless, and experienced another slight fit, the exploration was discontinued, and further exposure of the brain was made.

The animal now being quiet, the experimentation was recommenced.

Obs. 13.—Secondary at 6 cm.

Electrodes on point (13), fig. 6, the anterior extremity of the frontal division of the lower external convolution.

Twitching of the right corner of the mouth.

After several repetitions of this effect, a slight fit came on, limited to the muscles of the face and neck. After the cessation of this, in a few minutes another attack occurred without further application of the electrodes, in which violent twitching of the right ear was the most marked symptom.

Obs. 14.—Electrodes on point (14), fig. 6, a point in the same convolution just behind the last-mentioned.

Retraction of right corner of mouth, and drawing downwards and forwards of the right ear.

Longer application gave rise to spasmodic twitching of the right corner of the mouth and muscles of right side of neck. On another application a remarkable change in the aspect of the animal was induced. It began with wagging of the tail and spasmodic twitching of the right ear. After the cessation of the more violent spasms, the animal held up its head, opened its eyes wide with a most animated expression, and wagged its tail in a fawning manner. The change was so striking that I and those around me at first thought that the animal had completely recovered from its stupor. But, notwithstanding all attempts to call its attention by patting it and addressing it in soothing terms, it looked steadfastly in the distance with the same expression, and continued to wag its tail, for a minute or two, after which it suddenly relapsed into its previous state of narcotic stupor. It was evidently an acted dream.

Obs. 15.—Electrodes on point (15), fig. 6, a vertical gyrus just anterior to the sigmoid and continuous inferiorly with the mid-froutal.

Elevation of the right upper eyelid.

Obs. 16.—Electrodes on point (16), figs. 6 and 7, about the middle of the inferior exterual convolution (parietal region).

No movements were observed here. The animal became restless and uneasy.

Obs. 17.—Electrodes (secondary at 4 cm.), on point (17), figs. 6 aud 7, the posterior part of the upper division of the middle external convolution.

The auimal is uneasy and restless, crying and wagging its tail as if in paiu.

Experiments on this region were discontinued.

The hemisphere was then further exposed by extirpation of the eyeball, and removal of the orbital plate, &c.

Obs. 18.—Electrodes (secondary at 6 cm.), on point (18), fig. 6, towards the anterior extremity of the frontal division of the third or lower external.

Twitching upwards of right cheek, retraction of the corners of the mouth on both sides, and drawing downwards and forwards of left *side of the neck.* Whether the platysma or trapezius was in action, could not be satisfactorily ascertained. This experiment was repeated several times with the same result, and particular pains were taken to prove that the contraction of the cutaneous muscles on the *left* side of the neck was not due to conducted currents.

Obs. 19.—Electrodes on point (19), fig. 6, just posterior to last, on the same lower frontal convolution.

Drawing up of the right side of the face, and closure of the jaws. The right temporal muscle was strongly contracted, but on account of the reflection of the left temporal and removal of the coronoid process of the left ramus of the jaw, it could not be ascertained whether there was also siuultaneous active contraction of the left temporal muscle. The left ramus was, however, of course elevated by the action of the temporal of the right side.

Obs. 20.—Electrodes on point (8), figs. 6 and 7, as in Obs. 8.

The same effect as before, viz., closure of the right eye.

Obs. 21.—Electrodes (secondary at 8 cm.), on point (20), figs. 6 and 7, on the upper curve of the supra-Sylvian convolution just above the fissure.

No result observed.

Obs. 22.—Electrodes on point (21), fig. 6, on the supra-orbital convolution, just at the anterior extremity of the lower frontal convolution.

Drawing back of the head and opening of the mouth. The animal makes a feeble attempt at a cry or growl. The animal at this stage was very much exhausted. Repeated applications of the electrodes to this point and its neighbourhood, caused whining and growling noises, such as a dog makes when dreaming. These cries were uot uttered on application of the electrodes to other parts of the brain at this time.

Obs. 23.—Electrodes on point (22), fig. 6.

(Secondary at 4 cm. as excitability of brain is diminished.) Poiut 22 is in the supra-orbital region, a little in advance of point (21).

The animal opens its mouth, retracts the upper lips, and makes a sort of sniffing or snarling noise.

Obs. 24.—Electrodes on point (23), fig. 6, near the frontal extremity of the superior external convolution.

Head suddenly jerked downwards on the chest.

Obs. 25.—Electrodes on point (24), fig. 6, a point on the supra-Sylvian gyrus, just anterior to the fissure of Sylvius.

There is twitching of the right ear; but at this stage the animal was so exhausted that no further exact conclusions could be drawn.

Experiments were then made on the cerebral ganglia. The results are given below.

The duration of the experiment was between three and four hours.

As in the case of the cat, another experiment was made to ascertain whether symmetry existed in the brain of the dog, and for this reason the right hemisphere of another dog was exposed.

Here also the success was only partial, but the results obtained are sufficient to indicate complete bilateral symmetry.

EXPERIMENT VII.—The greater part of the right hemisphere of a small poodle was exposed.

The animal was narcotised with ether.

Obs. 1.—Electrodes (secondary coil at 8 cm.) on point corresponding to (11), figs. 6 and 7, *i.e.* on the parietal region of the middle external, posterior to (5), a centre for the eyelid.

The head is rotated to the left.

Obs. 2.—Electrodes on point midway between (11) and (17), figs. 6 and 7, further back on same middle external convolution.

Head is rotated to the left, and gradually, with longer continued stimulation, a position with the right ear up and the left ear directed downwards is assumed.

Obs. 3.—Electrodes (secondary coil at 7 cm.) on point corresponding to (12), figs. 6 and 7, near the recurved portion of the lower division of the middle external.

Rotation of head to left as before.

If the results of these three observations be compared with those obtained by stimulation of the corresponding points in the left hemisphere of the other dog, it will be seen that the present are more definite. It was noted that the animal during stimulation of those points was in a very restless condition, and frequently was thrown into an epileptic fit by the application of the electrodes. The results, however, are, so far as they go, quite in harmony with each other, and I should be inclined to regard the present as those which will normally

be obtained from stimulation in this region. I am not yet in a position to assert this on the grounds of further experiment, however, as two other dogs died before results could be obtained, and further experiments were interrupted for a time.

Obs. 4.—Electrodes on point between (5) and (11), figs. 6 and 7, at the posterior extremity of the mid-frontal, or anterior division of the middle external convolution.

The left eyebrow is elevated.[1]

This result was not obtained alone in the previous experiments, as the exact point had not been irritated.

Obs. 5.—Electrodes on point corresponding to (16), figs. 6 and 7, *i.e.* on the middle of the inferior external convolution (parietal region).

The left ear is drawn downwards and backwards.

No result had been obtained in the former case (Exp. VI.), owing to the animal's restlessness and depressed condition of the brain.

Obs. 6.—Electrodes on point corresponding to (5), figs. 6 and 7, the posterior extremity of the mid-frontal convolution.

Forcible closure of the left eye.

The result is the same as in Obs. 5, Exp. VI.

Obs. 7.—Electrodes again applied to points posterior to and corresponding to (16), *i.e.* the recurved portions of the inferior external convolution.

Movements of the ear frequently caused. Exact notes are, however, wanting.

The animal is in an exhausted condition.

Obs. 8.—The secondary coil is advanced to 6 cm., and the electrodes applied to the posterior parts of the brain, on their external and internal surfaces, but no results were observed. They seemed insensible to irritation, the other parts being still excitable.

The electrodes were then applied to the anterior and posterior extremities of the superior external gyrus. The electrodes were slightly sunk into the cortical matter. An epileptic fit followed, with rotation of the head to the left and erection of the tail.

The electrodes were then pushed into the right corpus striatum, when immediately the animal passed into rigid pleurosthotonus, the head approximating the tail, and both fore and hind-legs firmly flexed. This condition lasted so long as the irritation was kept up. The spasms were purely tonic in character.

The whole of the right hemisphere was then removed, with the results which will be detailed below (p. 78).

A comparison of the two experiments, VI. and VII., indicate, as I have said, the symmetry of the hemispheres, and if the results be compared with those obtained from stimulation of

[1] Possibly this may have been to some extent associated with the action in Obs. 5.

corresponding parts of the brain of the cat, a great degree of resemblance will be found to exist.

Compare together the regions included within (1), (2), (6), in the brain of the cat, with that included in (1) and (†) in the brain of the dog.

The movements of the paws are centralised here in both.

Compare the region included within the points (7) and (8) in the brain of the cat, with that included in points (4), (15), (7), (6), (5), (8) in the dog.

The movements of the eyelids and face are represented here in both.[1]

Compare points (9), (10), (11), (12), (13), (14) in the cat, with the corresponding region (11), (12), (16), (17) in the dog. The lateral movements of the head and ear are centralised here in both. Compare region (9) in the dog with the corresponding region (3) in the cat. This is the centre for the tail in dogs, and probably also the extensors of the hind leg. The experiments in cats did not, however, give the same definite results.

In the region included within (13), (14), (18), (19), and probably (20) in the dog, corresponding with the inferior frontal and supra-sylvian convolutions, as well as in the orbital extremities of these convolutions indicated by (21) and (22), we have almost a complete parallel with the similarly situated regions in the cat included in the letters (18), (17), (16), and (15). All the unilateral and bilateral movements of the mouth, tongue and jaws, along with certain muscles of the neck, are here centralised, the centres for opening the mouth being in advance of the fissure of Sylvius, while those which act in closure of the jaws are somewhat posterior and more in the region of the fissure itself. In the tip of the frontal regions as indicated by (19) and (20) in the cat, and by (23) in the dog, there are certain movements of the head and neck centralised in both, the exact signification of which, and their correspondence with each other, will require some further experimental elucidation.

Irritation of the posterior lobes of the brain, both on their external surface and on the internal or hippocampal surface,

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[1] The eyeballs also.

as well as irritation of the gyrus fornicatus always failed to produce outward manifestations. These regions, as well as the hippocampal convolution itself, seem to be more especially connected with the sensory tracts. There are many anatomical, apart from experimental, data for drawing such a conclusion, but I do not here propose to enter into the question at greater length. I would only call attention to the fact of the outer root of the olfactory bulb being traceable to the tip of the uncinate convolution in all orders of animals. And there are many clinical facts which tend to point to this region as more particularly connected with the sense of smell.

A consideration of this subject, however, will involve a much more extended investigation than I have yet been able to make, and I merely indicate a point to be attended to in clinical observation and morbid anatomy of the brain.

I have next to record the results of systematic experiments similar to those in the case of the cat and dog, with a view to localise the centres in the brain of the rabbit.

There is greater difficulty in this case as regards exact localisation on account of the absence of such landmarks as are afforded by the convolutions, and therefore I am obliged to indicate the regions by the marks in the woodcut, and only in a general way describe the position of the electrodes on the hemispheres.

As in the case of the cat and dog, I also made comparative experiments on the right and left hemisphere. The results here also possess the same degree of uniformity, allowing for all difficulties in the way of exact comparison.
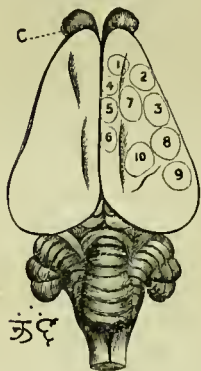
Fig. 8. — Upper surface of the brain of a rabbit. The letters have the same signification as in the preceding figures. The positions of the letters are described in the text.

EXPERIMENT VIII. — A half-grown rabbit. Right hemisphere exposed.

Secondary coil at 8 cm.

Obs. 1.—A preliminary application of the electrodes on point (1), fig. 8, with the secondary coil at 10 cm., produced no result. The secondary was then moved up to 8 cm. Momentary application to this point, the anterior part of the frontal region caused *twitching of the left upper lip, and slight*

drawing of the head to the left. Longer application caused decided drawing of the head to the left, and a munching movement of the lips and mouth, more particularly on the left side.

Obs. 2.—Electrodes on point (2), fig. 8, a little more to the side of the frontal region, situated just posterior to the centre of the orbit.

The mouth is drawn to the left, and a munching movement of the left side of the mouth is made, as if the animal is eating. Longer application causes the head also to be drawn to the left side.

Obs. 3.—Electrodes on point (3) a little further back. The same movements are induced as in the two previous observations.

Obs. 4.—Electrodes on point (4), fig. 8, parallel to the longitudinal fissure in the anterior region, where a superior external convolution is indicated.

Immediate elevation of left shoulder and extension of the toes. When the stimulation had been kept for a few seconds, the left paw and toes continued for some minutes in a state of choreic movement.

Obs. 5.—Electrodes on point (5), fig. 8, posterior to point (4).

Retraction, with adduction of left paw and extension of the toes.[1]

Obs. 6.—Electrodes ou point (6), fig. 8, still further back parallel to the median fissure.

While preparing for this experiment, the animal experienced a slight epileptic seizure, characterised by choreic convulsive spasms of the left lip and eyelid, with drawing of the head to the left. The fit lasted ten seconds.

On application, no very distinct results could be observed, as another epileptic fit came on similar to the preceding. (But see result of similar application in the succeeding experiment, IX.)

Obs. 7.—Electrodes on point (7), fig. 8, *i.e.* in the middle of the anterior triangle already marked off.

There are munching movements of the left upper lip and grinding of the jaws, as if the animal were eating vigorously. The head is also drawn to the left. Touching any point in this anterior triangle causes these movements of the lips and jaws.

After these observations, another fit, lasting 20 seconds, came on, similar in character to the two already mentioned.

Obs. 8.—Electrodes on point (8), figs. 8 and 9, a point described in my notes as above and posterior to the fissure of Sylvius.

Immediate forcible closure of the left eye.

(See Obs. 9, Experiment IX.)

Fig. 9.—A side view of the right hemisphere of the same brain as fig. 8. The letters and numerals have the same signification.

Obs. 9.—Electrodes on point (9), just behind point (8), produced same result, viz. *closure of the left eye.* Movements of the ear were not recorded in these observations. (But compare the corresponding observations in Experiment IX)

Obs. 10.—Irritation over the prominent portion of the parietal portion of

[1] This combined movement is just the same as described in the case of the cat and dog.

the brain, as indicated in point (10), figs. 8 and 9, gave no definite results which could be fixed on.

(But see corresponding experiment, Experiment IX.)

Obs. 11.—Electrodes on point (11), fig. 9, *i.e.* at the under and lateral aspect of the anterior or frontal lobe.

Munching of lips and opening and shutting the mouth.

Obs. 12.—Electrodes on point (12), fig. 9.

Similar movements of the lips and jaws, as if eating.

Obs. 13.—Electrodes pushed under the natiform protuberance (Owen), or tip of the hippocampal gyrus, produced no very distinct results; the animal started suddenly backwards, twitching its left ear, but whether this was the result of irritation of sensory regions of the brain, or of the dura mater, could not be satisfactorily determined.

The quite posterior regions gave no results on stimulation.

The other hemisphere was then exposed, and irritation applied; but the notes only indicate generally where electrodes were situated.

Obs. 14.—Electrodes applied to upper and anterior part of the left hemisphere.

Head is drawn to the right, and the animal makes munching movements of the lips on the right side.

The brain was then further exposed, and experiments on the corpora quadrigemina and ganglia were made. These will be detailed below.

In accordance with the plan of experiments on cats and dogs, I now give the results of an experiment on the left hemisphere of another animal, for comparison as to symmetry.

EXPERIMENT IX.—Medium-sized adult rabbit. Left hemisphere exposed.

Secondary coil at 8 cm.

Obs. 1.—Electrodes at point corresponding to (1), fig. 8.

Spasmodic elevation of the right lip, followed by munching movements of the lip and mouth.

Obs. 2.—Electrodes on points corresponding to (2) and (3), fig. 8.

Similar results, namely, retraction of right lip, and munching movements of mouth, more marked on right side.

After a few applications, the animal had a slight fit, in which the head was drawn to the right, and the right lip spasmodically twitched.

The results exactly corresponded to those recorded in Obs. 1, 2, and 3, Exp. VIII.

Obs. 3.—Electrodes on point corresponding to (4), fig. 8.

Immediate elevation of right shoulder, and extension of toes of right paw.

This result is also in complete harmony with Obs. 4, Exp. VIII.

Obs. 4.—Electrodes on point corresponding to (5), fig. 8, *i.e.* just posterior to (4), and parallel to the median fissure.

Immediate retraction, with adduction and flexion of right paw from the extended position.

This also is in complete harmony with similar Obs. 5, Exp. VIII.

Obs. 5.—Electrodes at point (6), posterior to (5), fig. 8, still parallel to the median fissure.

Distinct flexion, and advance of the right hind leg from its extended position.

It will be seen by comparison of this observation with Obs. 6, Exp. VIII., that the results in this case are more definite, due to the absence of the complications caused by the epileptic seizure in the first.

Obs. 6.—Electrodes again applied to anterior extremity of frontal lobe, on points corresponding to (3) and (7), figs. 8 and 9.

Again munching of lips and jaws, right lip always drawn up.

As the animal was becoming restless, it was narcotised with chloroform. During the narcosis, the right forepaw executed a most rapid series of movements. The movements were those of alternate extension flexion, and rotation from without inwards of the limb.

This subsided into more choreic-like twitches of the limb, and twitchings of the lips and whiskers on the right side were also observed to continue for some minutes. The movements were again intensified by the re-administration of chloroform, which had been discontinued when the movements first began.

When the animal had subsided into a quiescent state, experimentation was again proceeded with.

Obs. 7.—Electrodes on point corresponding to (12), fig. 9, anterior to fissure of Sylvius.

Opening of the mouth. Distinct bilateral action.

If Obs. 12, Exp. VIII., be referred to, it will be seen that the results are quite in harmony with each other, indicating a centre for the mouth and jaws.

The condition of the tongue was not observed.

Obs. 8.—Electrodes on point posterior and superior to (8), fig. 9, *i.e.* over the parietal region (10).

The head is turned to the left.

A distinct rotation of the head to the other side was not so definitely ascertained in the previous experiment, but it is in complete harmony with similar observations in the cat and dog, when the electrodes were applied on homologous parts.

Obs. 9.—Electrodes on point corresponding to (8), fig. 9, behind the fissure of Sylvius.

The eye is forcibly closed. The ear was also observed to close at the same time. Movements of the ear were always induced along with those of the eyelids, but there was considerable difficulty in defining their exact scope and range.

By referring to Obs. 8, Exp. VIII., it will be seen that the movement of the eyelids is recorded exactly the same as in this case.

Obs. 10.—Electrodes on a point just anterior to (8), fig. 9, and marked (13).

The eye is opened, and some movements of the ear are made, apparently a tendency to retraction.

No definite point of comparison with this exists in the previous experiment.

·The other experiments were all in harmony with those already recorded in Exp. VIII., the more posteriorly-placed parts being, as before, apparently insensible.

Subsequent investigation may enable me to define with greater accuracy the situation of the centres, but I think that the results given indicate with a tolerable degree of definiteness the regions for the paws, the eye, and the mouth and neck, as in the brains of the cat and dog.

The correspondence is striking, and yet there are very remarkable differences. It will be seen that there is a general correspondence in the situation of the centres for the paws, the eye, the mouth and jaws in the anterior parts of the hemisphere of the rabbit with the like centres in the hemispheres of the dog and cat, viz., from the median fissure down towards the orbital surface, paws, eye, and mouth.

The region (4), (5), (6), parallel to the median fissure, or, as it may be said, the superior external convolution in the rabbit's brain, corresponds in function, to (1), (2), (6) in the cat, and (1), (†), (9) in the dog.

In the cat the paw is most differentiated; in the dog the paws and tail, and in the rabbit the fore and hind paw.

Then again, (13), (8), and (9) in the rabbit correspond in function and in situation with (7) and (8) in the cat, and (15), (4), (7), (6), (5) and (8) in the dog, *i.e.* the movements of the eyelids are represented in all, with a varying degree of differentiation in each, in the mid-frontal region.

Associated with these, and including (10) in the rabbit, are the movements of the ear and muscles of the opposite side of the neck, comprised within a region which essentially corresponds in situation to (9), (10), (11), (12), (13), (14) in the cat, and to (11), (12), (16), (17) in the dog, *i.e.* in what may be termed the parietal region.

The movements of the lips and mouth are represented in regions at first sight, diverging considerably in situation from the corresponding regions in the cat and dog. It will be seen that not only in the lower frontal (11), (12), in the rabbit, but also to a very considerable extent in the superior

and anterior frontal regions, the movements of the lips and mouth are centralised. Those in the cat and dog are entirely confined to the lower frontal and orbital regions and neighbourhood of the fissure of Sylvius.

It would appear as if, with a more defective development of the upper anterior or frontal lobes in the rabbit, the lower frontal are more highly developed, and rise into a position in the anterior portions of the hemispheres corresponding in situation with the superior and mid-frontal regions in the more highly-developed brains of the cat and dog. If such be the true homology, the correspondence between the regions for the paw, the eye, the mouth, ear, &c., will be seen to be almost complete in all. And the comparatively higher development of the centres for the movements of the mouth in rabbits would be quite in accordance with the habits of the animal, just as we see the higher development of the centres for the paw in cats, and the tail in dogs.

Several experiments were made on pigeons both in reference to the artificial production of epilepsy and the localisation of motor centres, but, as I have already stated, I obtained no results, the brains of these animals being apparently insensible to electric stimulation. This I found to be the case not only on the surface of the hemispheres, but also when the electrodes were pushed deeply into their substance. The cerebellum both of pigeons and fowls I found likewise insensible to the electric currents.

For these reasons I am unable to say anything regarding the homologues between the various parts of the brain of birds and those of mammals from an experimental point of view. Further investigation may,. however, discover some method of artificially discharging the cerebral centres in these animals also.

In order to complete the exploration of the brain, experiments were likewise made in almost every case on the cerebral ganglia when the excitability of the convolutions had become too far depressed or irritable for further accurate investigation.

The results may be stated in brief terms without entering into detailed notes of each experiment.

The method adopted was rapidly to lay bare the brain sufficiently to allow of easy division of the corpus callosnm and opening of the interior of the cerebral ventricles, so as completely to expose the basal ganglia. The application of the electrodes to the corpus striatum in dogs caused during the irritation most rigid pleurosthotonus, the head approximating the tail, the muscles of the face and neck in rigid tonic spasm, and the fore and hind limbs fixed and rigidly flexed. The predominance of the flexors over the extensors was marked. Removal of the electrodes caused entire cessation of the spasm. There were no choreic spasms during or after the stimulation. Apparently every muscle, or group of muscles represented in the convolutions, along with the lateral muscles of the body, were stimulated to contraction from the corpus striatum. The elcctrodes were applied only to the surface without mechanical injury. The action is always crossed—irritation of the right corpus striatnm producing left pleurosthotonus, and that of the left, right pleurosthotonus. Shifting of the electrodes on to the optic thalamns on either side produced no resnlt. Re-application to the corpora striata again induced pleurosthotonus.

Application of the electrodes to the hippocampus or parts surrounding the descending cornu of the lateral ventricle failed to produce any outward resnlt.

In rabbits, on whom the experiments were most numerous, irritation of the corpus striatum always caused rigid rotation of the head to the opposite side, but general plenrosthotonns was not observed. Freqnently, also, along with the rotation of the head, the jaws were ground together, not being maintained in rigid tonic spasm.

Shifting of the electrodes from one corpus striatum to the other indnced corresponding crossed neck and jaw spasm.

Electrodes on the pes hippocampi as it lies on the optic thalamus, as well as on the hippocampns itself and the fornix, always failed to produce any outward manifestation. For the sake of comparison, the electrodcs were always shifted on to the corpns striatum with the usnal result.

Reflection of the pes hippocampi exposed the optic thalamus. Electrodes on this gave no rcsult.

From the absolute uniformity of the negative results obtained by stimulation of the optic thalamus, fornix, and hippocampus, I conclude that these parts have no motor signification. Nor can it be stated that the irritation caused pain, for the animals never gave outward manifestation of such sensations.

Whatever their exact function may be as regards sensation or the elaboration of sensations, it may be difficult to say, but that these parts, and especially the optic thalami, are not motor ganglia may be stated with the utmost certainty, if one may conclude from the simultaneous excitability of the motor ganglia and the absolute insensibility of the optic thalami themselves.

The method of exploration I have adopted is more likely to lead to accurate results than the older methods of laceration or excision. The irritation being applied only to the surface, and simply exciting the functional activity of the ganglia, excludes the possibility of injury to the motor strands passing through the optic thalami to the corpora striata. Without entering into a discussion of the views of previous experimenters on the cerebral ganglia, but for the present confining myself exclusively to the results of my own experiments, I should be inclined to attribute results of lesions 'destroying' or 'discharging' of the optic thalami, in so far as they induce paralysis of motion or the reverse, to affections of the motor strands of the cerebral ' projection system ' (Meynert) passing downwards from the corpora striata, and through and underneath the optic thalami to the cura cerebri. This view, in my opinion, would be quite in harmony with the facts of clinical observation, and, taken with the results of irritation of the motor centres, would explain the symptoms which have led Meynert [1] to diagnose disease of the optic thalamus during life.

Experiments as to the motor signification of the corpora quadrigemina, performed chiefly on rabbits, gave results of great definiteness. These ganglia are exceedingly sensitive to electric stimulation.

[1] Stricker's ' Med. Jahrbücher,' 1872, p. 188.

My notes of the first experiment made on these ganglia in a rabbit are as follows :—

The electrodes (secondary coil at 5) applied to the anterior tubercles of the corpora quadrigemina caused immediate and violent opisthotonic spasm, the head and tail being curved rigidly backward, the fore and hind legs rigidly extended. The pupils were observed to be widely dilated, and the jaws firmly clenched.   A second application of the electrodes caused such violent and sudden recurrence of the opisthotonus that the electrodes were driven into their substance, as was afterwards ascertained, to the extent of two lines.

On this occasion the pupils became contracted, a result which I attributed to irritation of the nuclei of the third nerves, situated just below the aqueduct of Sylvius, at the point where the injury was caused.

The opisthotonic spasms were induced on each application, and were of the most severe description, lasting generally nearly half a minute after the withdrawal of the electrodes.

Another experiment, made specially to determine whether the corpora quadrigemina exercised crossed action on the extensor muscles, gave me the following results :—

Irritation of the right anterior tubercle caused dilatation of both pupils (whether the left more so than the right was not noticed), and extension of the body and legs, the left much more so than the right.

Irritation of the left tubercle caused exactly opposite results.

Stronger irritation of one or both caused general opisthotonus, as in the previous experiment.

Hence it would appear that the action of the corpora quadrigemina is more or less crossed, but that violent irritation is sufficient to stimulate the extensors of both sides.

These experiments on rabbits I have repeated many times, and always with the same results.   Indeed, so immediate is the opisthotonus, that very considerable care and dexterity are necessary to avoid injury to these ganglia by the electrodes, owing to the sudden manner in which the head is thrown backward. The jaws were always violently clenched.

If the animal is not tied down it is merely necessary to touch the corpora quadrigemina with the electrodes to make it execute a backward somersault head over heels, and throw itself off the table. In experimenting on the cerebellum, especially on its anterior surface, it is absolutely requisite to keep the electrodes insulated and sufficiently distant from the corpora quadrigemina, if the results are not to be complicated and the experiment spoiled by the sudden opisthotonus which irritation of these ganglia induces unless the head is firmly fixed.

In an experiment on a cat, after I had been exploring its convolutions and exhausting their excitability, I pushed one of the electrodes down between the hemispheres posteriorly, keeping the other on the anterior surface of the brain. Immediately the head was thrown back in rigid spasm, the jaws firmly clenched, and the angles of the mouth retracted to their fullest extent in the most typical form of *risus sardonicus*. I at once concluded that my electrode was on the corpora quadrigemina, but kept it in its position, and moved the other to different parts, merely to open and close the circuit. Each time the circuit was closed the same result followed. Keeping the electrode fixed in its position, I dissected down, and found it placed almost on the central point between the nates and testes on each side, without laceration of their substance.

The general condition of opisthotonus was not so well marked as in the cases of the rabbits, but I attributed the results to the already greatly depressed condition of the ganglia at the time.

On two other occasions I attempted to expose the brain and dissect directly down on the corpora quadrigemina in the cat, but failed to get them clearly exposed for experimentation before the death of the animal, or at least complete insensibility of its cerebrum.

Ultimately I succeeded in exposing the nates and testes of the left side. Irritation of both ganglia caused firm clenching of the jaws and retraction of the angles of the mouth. I could not localise the irritation on one or other distinctly, however. On strengthening the current, along

with trismus, violent opisthotonus came on, with extension of the limbs, retraction of the head, and elevation of the tail.

The results were therefore in complete accord with those in rabbits. Trismus seemed to be the first stage in the progress of general opisthotonic spasm.

These results may throw some light on the true pathology of tetanus with opisthotonic spasm; and it will be interesting to examine the condition of the corpora quadrigemina in death in this state.

In connection with these results of electric stimulation, I record an experiment made on a pigeon, which I at first was unable to explain, not having at the time a clue to its interpretation.

After endeavouring in vain to excite movements by faradisation of the pigeon's hemispheres, I completely extirpated both hemispheres. The animal was immediately seized with opisthotonus, the neck being recurved till the beak pointed straight upwards, the tail curved backwards, and forming almost a complete arch with the head; the legs were extended, and the wings slightly drawn upwards and backwards. The tetanic spasms occasionally remitted, but there were no complete intermissions. Any such irritation as a jar, a touch, &c., immediately intensified the tetanic condition.

The animal was then killed, and the condition of the brain examined. The cerebral hemispheres were found to have been completely removed. The optic nerves were uninjured, but there was laceration of the anterior surface of the optic lobes, more marked on the right side, where the optic tract had been peeled off to some extent. I have not made any further experiments on the optic lobes of birds, nor have I tested their irritability by the electric method; but the results here given seem to receive a satisfactory explanation from the vital irritation of these ganglia, set up by the mechanical injury which they had suffered. The continuance of the tetanic spasms would also point to this injury as being the cause of the permanent irritation.

The structural homology subsisting between the optic lobes and the corpora quadrigemina, on which I have made extended histological research, will be the subject of a future

paper, but the result of the experiment just narrated may be taken as, to some extent at least, corroborative of the physiological homology of these ganglia.

That the integrity of the corpora quadrigemina is essential to vision, is a well-ascertained fact (see below, p. 78), but it is not by any means satisfactorily determined whether the loss of vision caused by destructive lesions of these ganglia is due to the destruction of terminal or only intermediate centres in the course of the optic tracts. The intimate relation subsisting between the optic lobes and cerebellum in all animals, and more particularly so in the lower orders of the vertebrata, taken with the results of experiments on the cerebellum detailed below, indicate facts of very great significance with regard to the cerebellar relations of the optic centres.

And while it may be, I think, fairly concluded from my experiments that the corpora quadrigemina are centres, intermediate or otherwise, for the extensor muscles, I would not have it understood that I regard this as an exhaustive account of the functions of these ganglia. My experiments on them are not yet sufficiently extensive to allow of my making precise statements as to what other signification they may have, and therefore I reserve for further experimental investigation a consideration of these points.

Before summing up or generalising from the results of the experiments detailed above on the cerebral hemispheres and ganglia, I now proceed to tabulate the results of experiments on the cerebellum.

To expose completely and carry out the same definite system of exploration on the cerebellum as I have applied to the cerebral hemispheres and ganglia, is a matter involving very considerable difficulty. Like the hemispheres, the cerebellum is susceptible to faradic stimulation, but, as in them, the excitability is rapidly exhausted by profuse hæmorrhage. Operations to lay bare the cerebellum necessarily involve profuse hæmorrhage, from the numerous venous sinuses meeting at the internal occipital spine. For this reason I found it almost impossible to carry out complete

exploration of the various lobes and lobules in any one
animal. My experiments, therefore, had to be extended
over a great many different animals, in order to determine
with accuracy the functions of those parts which could not
be clearly defined in the one or other instance.

In many cases, however, a tolerably complete exposure
and exploration of the whole cerebellum could be carried
out. As in the case of the hemispheres, I found that the
hæmorrhage could often be very effectually controlled by the
insertion of cotton wool into the bleeding orifices. My first
experiment was made on a guinea-pig, and the cerebellum
was only so far exposed, by trephining out a small circlet
of bone on each side of the occipital protuberance sufficient
to allow of the application of the electrodes to the surface of
the cerebellum directly.

The results were such as to lead me at first to suppose
that the cerebellum was not excitable, or, at least, if so it had
no special motor signification. With and without chloro-
form, the application of the electrodes apparently produced
no effect, the animal remaining perfectly quiet. Occasionally
during application of stronger currents, the animal exhibited
restlessness, and uttered cries, but they did not indicate
anything further than the usual restlessness and cries of
guinea-pigs when under experimentation.

With the secondary coil at 4 cm. the electrodes were
applied for one minute without any apparent result. The
electrodes were applied as gently as possible to the surface,
but some slight laceration of the substance was accidentally
made.

The animal, after the irritation, could preserve an upright
but rather unsteady position on its feet; but when it at-
tempted to walk it reeled from side to side, and fell repeatedly
in a very intoxicated like manner. The eyes were not exa-
mined.

The animal was then killed, and the cerebellum examined.
The electrodes had been applied to the upper and anterior
parts of the lateral lobes, and laceration to the depth of a
line had been made in the cortical substance. The result
was therefore only a confirmation of the every-day experi-

ment, proving that lesions of the cerebellum cause a want of co-ordination of the muscular action necessary to maintain the equilibrium of the body. The next experiment, made on a rabbit, in which, after experimentation on the basal ganglia, the cerebellum was subjected to electrical stimulation, yielded results which led to a more elaborate series of experiments, demonstrating the fact that the cerebellum co-ordinates the movements of the eyeballs.

Repeated applications of the electrodes to the cerebellum were chronicled in the notes as producing no results, but after continued application to the anterior parts of the lateral lobes, I observed that the eyeballs were affected with violent nystagmus, and subsequent application of the electrodes to different parts of the cerebellum distinctly showed that movements of the eyeballs in different planes were produced by stimulating different points. The pupils were stated in the first notes as becoming contracted with each stimulation, but attention was afterwards so exclusively devoted to the movements of the eyeballs themselves that the condition of the pupils is not recorded.

Without entering into the results obtained from each animal experimented on, I include under the head of one experiment the general results of experiments more or less complete on twelve rabbits. The difficulties in exploring the various parts of the cerebellum are, as I have said, much greater than in experiments on the cerebrum. In addition to the dangers from hæmorrhage, the experiments are complicated by the fact that the movements of the eyeballs are less easily noted than those of the limbs, and frequently, after prolonged irritation of one part in order to verify the first results, a condition of nystagmus comes on, and the movements presided over by the other parts are complicated by the epileptic condition of the already irritated centres. When too strong currents are used, necessitated by the depressed excitability of the cerebellum, movements may be induced which are not strictly due to localised irritation. In addition, also, the position of the animal's head, and its condition as to anæsthesia, seem to modify the results. Hence some analysis is necessary, and the various conditions

I have mentioned must be borne in mind by those who wish
to repeat my experiments.

I give the following results as those which I have most
constantly obtained, but I do not state them so definitely as
not to require further verification, and perhaps more accu-
rate description, with further analysis of the ocular muscles
concerned in the various movements.

EXPERIMENT X.—Rabbits.   Cerebellum exposed by successive removals
of the occipital bone.   Dura mater stripped off.   Secondary coil usually at
8 cm.   Electrodes applied to the points indicated in figs. 10 and 11.

Obs. 1.—Electrodes at (1), fig. 10, on the upper division of the middle
lobe of the cerebellum.

The right eye moves outwards and the left inwards in a horizontal plane.
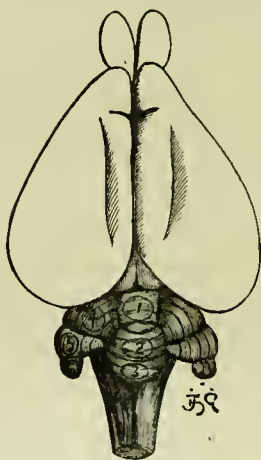
Fig. 10.—Upper surface of the cerebrum and cerebellum
of rabbit. The numerals (1), (2), (3) are on the upper,
middle, and lower divisions of the median lobe of the
cerebellum.   The numerals (4), (5), and (6) are on
the upper, middle, and lower divisions of the left
lateral lobe.

Fig. 11.—Anterior aspect of same
cerebellum, cut off just pos-
terior to the corpora quadri-
gemina. E is on the cut surface
of the pons Varolii. Numeral
(7) is on the anterior flocculus
of the lower division of the left
lateral lobe. Numeral (8) is on
the anterior aspect of the upper
division of the same lobe.

Owing to the position of the eyes in the rabbit these movements might
be described as backwards and forwards, instead of outwards and inwards.

Obs. 2.—Electrodes on middle of middle lobe of cerebellum, point (2),
fig. 10.

*The right eye moves inwards and the left outwards in the same horizontal
plane.*

Obs. 3.—Electrodes on lower division of middle lobe, point (3), fig. 10.

The same movements are repeated as in Obs. 2, viz. *the left eye outwards,
the right inwards.*

It would thus appear that the upper and lower divisions of the middle
lobe reverse each other's action.

The middle lobe was explored in three points, because, as will be seen in the figure, it is apparently divided into three divisions. There is not, however, a physiological division.

In making these experiments the head of the rabbit was held in such a position as to make the angles of the eyes as nearly as possible horizontal, so that the movements are described in reference to this plane. It is very necessary to localise the electrodes exactly on the centre of the lobules, and not approach too near the lateral lobes, otherwise contradictory results will be got.

Obs. 4.—Electrodes on posterior surface of the upper division of the left lateral lobe, point (4), fig. 10.

The right eye moves downwards and rotates backwards or outwards, the left eye moves upwards and rotates forwards or inwards.

Occasionally, from electrisation of this lobule, I have observed the reverse of this movement, viz. upwards and forwards of the right eye, and downwards and outwards of the left; but the other seems, from numerous observations, to be the primary, and the second I attribute to a sort of reflex oscillation. Sometimes this oscillation, or nystagmus, would continue for some time.

Irritation of the corresponding upper division of the right lateral lobe reverses the action of the left, making the right eye move downwards and outwards, and the left upwards and forwards.

Conjoint irritation of both upper divisions neutralises both effects.

Obs. 5.—Electrodes on middle division of left lateral lobe, point (5), fig. 10.

The right eye moves downwards and rotates from without, inwards, in a direction contrary to the hands of a clock; the left eye moves upwards, and rotates, from within, outwards, in the same direction as the hands of a clock.

Obs. 6.—Electrodes on lowest division of the left lateral lobe, point (6), fig. 10.

The right eye rotates on its antero posterior axis, from within, outwards, the upper end of the vertical meridian moving contrary to the hands of the clock; the left eye rotates on its own axis, from without, inwards, the vertical meridian inclining in the same direction. The vertical meridians maintain their parallelism.

Obs. 7.—Electrodes on the anterior flocculus of the lower division of the left lateral lobe, point (7), fig. 11.

The right rotates on its own antero posterior axis, from without, inwards, the vertical meridian moving with the hands of the clock; the left could not be observed.

I have only as yet been able to obtain this result once, as the difficulties of reaching this portion of the cerebellum are great, and therefore it may stand in need of further verification.

The lower divisions of the lateral lobes seem, therefore, the centres for the rotation of the eye on its antero posterior axis.

Obs. 8.—Electrodes on the anterior surface of the upper division of the left lateral lobe, point (8), fig. 11.

Both eyes move upwards, and then downwards. An upward and downward oscillation generally ensued on irritation of this surface, and lasted some seconds.

I have not been able to localise more definitely the centres for the downward and those for the upward movements.

A condition of nystagmus is, as I have already stated, a frequent result of prolonged irritation of the cerebellum.

In my notes I find that sometimes irritation of the cerebellum, but whether locally or not is not stated, caused protrusion of the eyes and great convexity of the cornea.

Another fact noted, but whether causally related with irritation of the cerebellum or not remains to be discovered, is, that along with the movements of the eyeballs, dilatation of the nostrils, and a backward movement of the whiskers were frequently seen.

Further than that electrisation of the cerebellum of the cat causes movements of the eyeballs, as in the rabbit, I have not as yet localised the individual centres in other animals.

Many of the results recorded in the foregoing pages carry in themselves their own explanation, and require little further interpretation of their physiological signification.

The method of experimentation which has been followed may be regarded as one adapted for the artificial development of the function of the various parts of the encephalon by exciting in them a condition similar to that which in the normal order of events accompanies or coincides with psychical or volitional stimuli.

Cerebral activity, like the activity of every bodily organ, is associated with an active condition of the circulation. The electrical stimulus (whatever other effect it may have on the nerve cells) excites in the cerebral hemispheres, locally or generally, that condition of hyperæmia which is the normal physiological condition of an organ functioning actively. So far, therefore, moderate stimulation seems to excite no abnormal action, and the muscular movements called forth by the irritation of the individual convolutions may be regarded as the artificial excitation of their normal functional

activity. It is this positive character of the results of this method that renders it such a valuable instrument in the investigation and localisation of cerebral functions. But the movements evoked by stimulation of the hemispherical centres have a far deeper signification than the similar movements which may be caused by direct stimulation of the muscles themselves, or their motor nerves. It will be seen that the movements recorded in the above experiments as resulting from excitation of the individual centres are purposive or expressional in character, and such as we should, from psychological analysis, attribute to ideation and volition if we saw them performed by others. The clutching or striking movement of a cat's paw is not a simple muscular contraction, but is a complex and combined action of numerous muscles all directed to one end. Of course we have no other guide than our own consciousness to the interpretation of the actions of the lower animals, but as in ourselves or others we attribute such apparently purposive complex movements to ideation and volitional impulse, we may conclude that the cortical centres are not merely motor but voluntary motor, and concerned with the outward manifestation of intelligence.

The important question next arises, What is the relation between the convolutions as motor centres, and the parts of the cerebral hemispheres which are subservient to the mental processes more immediately related to these definite muscular movements? Are the ideational centres situated in the same regions as the corresponding motor centres; or does a high development of certain motor centres indicate only, but without localisation, a corresponding development of the ideational centres which manifest themselves outwardly through these?

Would a high differentiation of the centres for the hand indicate a high degree of manual *habilité,* or at least the capacity of acquiring such, and the possession of those mental powers more immediately associated with such dexterity.[1]

[1] In connection with this speculation I would refer to the words of Herbert Spencer:—'All that we need here notice is the extent to which in the human race

These speculations are suggested by the now tolerably well established fact of loss of speech following destructive lesions of the lower frontal convolutions in the neighbourhood of the Island of Reil. I say lower frontal convolutions without specially designating them as on the left side for reasons specified below. It is a significant fact that the centres for the mouth and tongue in cats and dogs are localised in regions corresponding in geographical position, and which, both anatomically and physiologically, I should be inclined to regard as the homologues of the lower frontal and Island of Reil in man. The question, then, is, do lesions in this neighbourhood destroy the organic centres of the memory of words, or do they only interrupt the channels whereby these are manifested outwardly as articulate speech? The fact that the speechless patient is likewise unable to write, *i.e.* in the sense of expressing himself by written symbols, even though the hand may retain the power of tracing these, would seem to indicate that it is not the mere channel for the articulate expression of ideas that is interrupted, but that the very centres of word-memory are destroyed.

' It is *à priori* incredible that a person who cannot speak should be able to write. For when we write we merely translate nascently revived words into written symbols. . . . The fact that an aphasic person cannot write . . . . is the same defect as loss of speech in another manifestation. The speechless man cannot read, not even to himself. It is not that his eyes, or rather the parts of the brain which contain processes for the recognition of images of things, are affected, for he does recognise objects, and, when he cannot read, can recognise headings . . . . and knows by the hand-writing where a letter comes from. The difficulty is still one of loss of speech. Written and printed words strictly

a perfect tactical apparatus subserves the highest processes of the intellect. I do not mean that the tangible attributes of things have been rendered completely cognisable by the complex and versatile adjustments of the human hands, and that the accompanying manipulative powers have made possible those populous societies in which alone a wide intelligence can be evolved—I mean that the most far-reaching cognitions, and inferences the most remote from perception, have their roots in the definitely combined impressions which the human hands can receive.' (' Principles of Psychology,' 2nd edit. vol. i. p. 359.)

have no meaning. They are merely arbitrary signs of words. They require translation into words, and into an *order* of words. This translation the speechless man cannot effect, as he cannot revive words. He understands what we say to him, as, though *he* cannot revive words, the sounds of our words revive the motor symbols of words in his brain—*i.e. in the undamaged side* (these italics are ours). For the speechless man is not wordless; his defect is that he cannot revive words *voluntarily.*' [1]

Before, however, asserting positively that the organic centres of word memory are situated in the same convolutions as those which direct the movements of the tongue and mouth, we require to consider another hypothesis which may be advanced. It may be said that the centres for word memory are situated in some other part of the brain, but that internal revivication is only possible through the conjoint action of the articulating motor centres. Destroying lesions of these centres might thus cause loss of speech, external as well as internal, by interrupting one part of the conjoint process. And this hypothesis would receive confirmation from the fact that uneducated people in general really make the movements of articulation when reading and thinking, and a large proportion of the intellectual class make the same movements of the tongue and lips unconsciously. That thinking may go on without these outward manifestations would not, according to this hypothesis, prove that the articulating centres are not in a state of functional activity, but that they are inhibited or somehow merely thrown out of gear with the motor nerves and muscles. I should, however, be inclined to regard the intimate relation subsisting between ideation and the unconscious outward expression of the idea in muscular action as a strong proof of the close local association of the ideational and voluntary motor centres. Hence I should incline to the opinion that the organic centres of word memory are situated in the same convolutions as the centres which preside over the muscles concerned in articulation. If this be

[1] 'A Study of Convulsions,' by Dr. Hughlings Jackson (Reprint from the ' Trans. of the St. And. Med. Grad. Assoc.'), 1870, p. 21.

so, then we ought to have a hand memory, a face and eye memory, an ear memory, and thus we may ultimately be enabled to translate into their psychological signification and localise phrenologically the organic centres of various mental endowments. This I put forward only as a speculation, but numerous other facts which might be mentioned in connection with the concomitants of epilepsy give to the idea a certain degree of colour. From the experiments related there is every reason to conclude that the two hemispheres are symmetrical. If this be so it would be unphilosophical to say the third *left* frontal convolution is the seat of the faculty of articulate speech. We have seen that the hemispheres have, as a rule, crossed action. But this crossed action only extends to muscles, and combined muscular movements which act independently of each other. Obviously, in regard to the movements of the mouth and jaws, unilateral action, at least complete unilateral action, is impossible. And we find in the lower frontal regions centres which regulate the opening and closure of the mouth, the tongue, and the accessory muscles on *both* sides. Hence it is that destruction of the centres for these in one hemisphere does not cause paralysis of the muscles of articulation. The pathology of aphasia is quite distinct from that of the paralysis of Duchenne. It may be that the action of these muscles is to some extent weakened, but there is no obvious paralysis, and they are quite competent to perform their other functions in regard to mastication and deglutition. I agree with those who regard the hemispheres as of unequal preponderance in the initiation of voluntary movements. Just as most people are right-handed, and therefore ' left-brained,' so, it may be argued, the left hemisphere is the 'driving side.' The occurrence of aphasia with lesion of the left lower frontal is therefore to be explained by anatomical, or physiological peculiarities, which favour the occurrence of the pathological lesions here rather than in the corresponding right region, and by the fact that in most people the left hemisphere is the one which is usually selected, or selects itself, in the performance of voluntary acts. The occurrence of aphasia with lesion of the right side in left-handed people

would completely agree with this. Though the left may be the driving side, the right, without doubt, and possibly through the agency of the corpus callosum, becomes the seat of an organic memory corresponding in every respect to the acts done by the left. Hence the loss of speech remains only so long as it takes for the right hemisphere to become educated, and take the lead where formerly it only followed. During this interval the patient can only utter such expressions (like oaths and interjections) as have become automatic, and which have been taken out of the region of ideation and purely voluntary impulse.

The fact that extensive lesions, particularly if slowly developed, of the hemispheres on one or other side may exist without causing any obvious loss of voluntary movements, does not to my mind conflict with the positive results ascertained by the direct rousing of the functions of the various centres by means of electrical stimulation. Sudden lesions of a destructive character are accompanied by hemiplegia, which, though incomplete and usually of transitory duration, confirm by their negative phenomena the positive results of the other method. Fritsch and Hitzig ascertained that destruction of the centres, in which they had localised certain movements of the paw in dogs, caused a partial paralysis of the muscles set in action by galvanisation of the same centres.

In confirmation of these same results, I observe in my notes of experiments made on a rabbit, already alluded to (p. 36), that after about two-thirds of the superior surface of the left hemisphere had been exposed, the animal ultimately recovered, but the whole of the exposed part became the seat of suppuration and fungus cerebri; so that the greater part of the cortical substance was rendered entirely functionless. During the five days intervening between the first operation and the second, which necessitated its death, the animal had apparently recovered perfectly without any distinct paralysis of the opposite side, but it had lost its natural sense of timidity, and regarded things which at other times would have frightened it, with a degree of stupid unconcern. It was also observed that though there was no distinct paralysis

of the right side, the animal always had a tendency to run in a circle towards the right, as if the muscles of the left were acting more powerfully than those on the right side.   There was evidently distinct weakness of the right side, rendered more apparent by the preponderant action of the muscles of the left when the animal was made to move forward.

But the facts of the following experiments on dogs, at first sight seem hardly in accordance with the idea of crossed action in the cerebral hemispheres.   In two dogs, one of which has already been alluded to (p. 54), after failing to get further results from stimulation of the convolutions, I completely extirpated the right hemisphere.

I give the particulars only of one case, though the two were somewhat similar.   The first animal (p. 54) lived three days after the operation.   In the second I removed the whole of the right hemisphere just anterior to the corpora quadrigemina. A few minutes after the operation the animal began to howl and bark, the movements of the jaws not having suffered the slightest degree of paralysis.   The left side and limbs were distinctly weakened, while the right side and limbs seemed to retain their full power.   The animal frequently turned its head to the right and struggled with its legs as if to rise and walk.   The right fore and hind legs moved vigorously, but the left hind and fore legs were also frequently moved in a similar manner, though less powerfully.   The movements of the left legs alternated with those of the right, but occasionally when the right legs were held so as to get rid of their driving or leading action, the left fore and hind legs would be moved by themselves in the struggles to get free.   Thinking that possibly the corpora quadrigemina might be concerned in these bilateral movements, I broke up these ganglia on the right side.   But the phenomena remained much the same, though the animal seemed blinded, as it ran against furniture, walls, and jammed its head into impossible corners.   It retained the power of opening both eyes, and of howling and barking in a very vigorous manner. Apparently it remained quite conscious, for, when called to, it would struggle to get up, and would sometimes regain its feet, and even succeed in walking a few steps, when it would fall

over in a helpless manner. In these attempts the weakness of the left fore leg was very evident. An hour and a half after the operation, when in any way disturbed it made struggles to get up. In these the hind legs moved alternately, the right certainly more actively, but the left fore leg was scarcely moved, though the right retained full power. The animal growled and barked very frequently.

In order to determine whether the combined movements were conditioned by the voluntary impulse of the left hemisphere, I next proceeded, two hours after the removal of the right hemisphere, to expose the sigmoid gyrus of the superior external convolution of the left hemisphere. Having ascertained by electrisation that I could induce the usual movements of the right fore leg by stimulation of its centres here situated, I cut away the greater part of this gyrus, checking the hæmorrhage with cotton wool steeped in perchloride of iron. After this the animal ceased to struggle, and lay in whatever position it was placed. Pinching the toes caused reflex movements in all the four limbs, and at the same time the animal barked energetically and howled when pinched. Pinching of the tail especially caused the animal to bark. This condition continued for several hours, barking being always elicited and some reflex movements of the legs, but not to any great extent. The barking may also have been a reflex phenomenon, but from the fact that barking alone was sometimes induced without any marked reflex movements of the limbs, I was rather inclined to attribute the phenomena to retention of consciousness and distinct sense of pain. Ultimately (five hours after the first operation), no barking was caused, but only reflex of the limbs and trunk when the legs or tail were pinched. The dog survived for eight hours after the removal of the hemisphere.

On *post-mortem* examination it was found that the right hemisphere had been completely removed by the crus just anterior to the superficial origin of the third nerve. The other hemisphere, with the falx and all the cranial nerves on both sides, were intact. The corpora quadrigemina were found completely lacerated and broken up on the right side, and there was some effusion into the anterior tubercle on the

left. The sigmoid gyrus corresponding to points (1) and (2), fig. 7, and the superior external convolution just anterior to point (9) had been completely removed.

Otherwise the hemisphere was intact, there being only slight extravasation on the surface at its anterior extremity.

These phenomena both in the rabbit and dog have a very important bearing on paralyses depending on lesions of the hemispheres and corpus striatum in man. They seem not in accordance with the idea that the hemispheres exert crossed action, such as indicated both by stimulation of the cortical centres, and also of the respective corpus striatum. The facts are quite in accordance with Dr. Broadbent's hypothesis, that associated movements of both sides of the body are bilaterally co-ordinated in each cerebral hemi-sphere.[1]

According to this hypothesis, the movements which are most independent of those of the opposite side are most paralysed by destructive lesions of the hemispheres and ganglia. Thus the arm is more paralysed than the leg, be-cause the leg acts less independently of its fellow on the opposite side. In the same category as the leg Dr. Broad-bent places the muscles of the chest, as well as those of the neck and back. These latter, however, I would remove out of consideration, as well as the muscles of the eye-balls, because, as we have seen, they have centres in the corpora quadrigemina and cerebellum independently of the hemi-spheres. Restricting ourselves, therefore, to the movements of the face and limbs, it seems to me that the above facts are completely in accordance with a *physiological* bilateral co-ordination, though not perhaps a strictly anatomical one, at least as far as the highest centres are concerned. Stimu-lation of the cortical centres, it will be noted, caused move-ments in the face and paws only on the opposite side. And stimulation of the corpus striatum caused pleurosthotonus, the tonic spasm affecting only the opposite side. It must be remembered, however, that between the ' projection sys-tem ' of the highest centres and the individual motor nerves and muscles, there are lower centres, where many or all the

[1] ' Med. Chir. Review,' April, 1866.

muscles are again co-ordinated, probably in a different manner and for different purposes in each. Thus the muscles concerned in respiration are co-ordinated in the medulla oblongata, but many of these are again represented in the corpora quadrigemina, corpus striatum, and also in the hemispheres. Taking the muscles of the limbs, we find them co-ordinated in their spinal centres; many of their muscles are again co-ordinated, possibly for some other purpose, in the corpora quadrigemina; all are co-ordinated in the corpora striata, and all again in the hemispheres. A consideration of these points will, I think, enable us to account for and distinguish the kind and degree of the paralysis observed in lesions of various parts of the motor track. A lesion confined to the cortical centres causes only partial and transitory paralysis. Lesions of the corpus striatum cause more complete paralysis of the same muscles. The various fibres of the highest motor centres all converge into, and are again differentiated in these ganglia. It is reasonable to suppose that these ganglia retain an organic memory of the stimuli proceeding primarily from the hemispheres, and may carry out automatically the movements initiated by them.

Hence, and probably by the influence of the sound hemisphere when the other is the seat of lesion, the corpus striatum will carry out without difficulty the movements which are physiologically combined with those of the opposite side, and less easily those which have been least associated together. The movements of the leg will therefore be more easily carried out than those of the arm, and some of the movements of the arm more easily than others of a highly complex and independent character.

We must not compare in this respect the paw of a dog with the hand of man, for the dog possesses few movements of his paw which are not associated with those of the opposite side. Lesions of the corpus striatum, on the other hand, will cause loss of voluntary, and to a great extent of the sub-voluntary or automatic movements. But it is quite possible that at the stimulus proceeding from one hemisphere and one corpus striatum, the organic memory of the

lower centres, such as the spinal, will enable them to carry out on both sides the movements which have usually been physiologically combined.

Hence, as we have seen in the dog, the posterior extremity will retain its power for alternating and consentaneous action with the other, while the more independent fore leg will be almost, if not entirely paralysed. As to the mouth and tongue, such movements as are not only physiologically, but also anatomically co-ordinated in each hemisphere, will remain unaffected, while the others in the face, which are only physiologically combined, will suffer. But when the highest centres in the other hemisphere are also destroyed, as in the case of the dog, voluntary motion is no longer possible in the muscles co-ordinated there, and the corresponding muscles of the other side which could still act in association with these, even when their own higher centres are destroyed, become completely powerless. Reflex action will, however, still continue in all the limbs, so long as their spinal centres and nerve trunks are intact.

This method of explanation seems to me to accord with the experiments I have related, and with the facts of clinical observation in man.

What relation subsists between the hemispheres and the other cerebral ganglia is a more difficult question to answer. I have already shown that electric stimulation of the optic thalami fails to produce any outward movement, and that in all probability paralyses of motion from lesions of these ganglia are to be attributed to affections of the motor strands passing through them to the crura. The fact that excitation of the surface of these ganglia does not cause muscular contraction, while similar excitation at the same time of the surface of the corpora striata and corpora quadrigemina causes movements, is sufficient proof of the localisation of the irritation, and that the movements produced by irritation of the corpora quadrigemina are not to be attributed to conduction of the excitation to the motor strands beneath.

Whether the optic thalami are represented in all parts of the hemispheres, or only in certain regions, is a question not to be answered definitely by the results of irritation. The

more immediate connection of these ganglia with the hippo-campal convolution by means of the fornix is of significance, taken with the fact that these parts are like the optic thala-mus itself, unsusceptible to faradisation as far as outward movements are concerned.

But until I have had an opportunity of making further ex-periments I forbear to make any very definite statements as to the exact functions of these ganglia and their hemispherical relations. Nor do I as yet profess to be able to explain the true significance of the corpora quadrigemina in reference to the extensors, and their relations, if any, to the cerebral hemispheres. The muscles which irritation of these ganglia set in action are undoubtedly under the control of the will, and many of them—*e.g.* the muscles which produce trismus— are also represented in the cerebral hemispheres. In no in-stance, however, did stimulation of the hemispheres excite the extensor muscles of the back, which irritation of the cor-pora quadrigemina set into such violent action.

The corpora quadrigemina are in more immediate relation to the cerebellum, but whether these ganglia are subordinate to higher centres situated in the cerebellum is a point on which it would be premature to speak definitely. Occasion-ally it appeared during the course of experiments on the movements of the eyeballs as if irritation of the cerebellum had a tendency to cause opisthotonus, but I rather attri-buted the phenomena to some degree of irritation of the corpora quadrigemina themselves. The spinal and cerebellar relations of these ganglia are of interest in reference to the muscles they set in action, and to the functions of the cere-bellum as a co-ordinating centre for the muscles of the eye-balls.

The corpora quadrigemina (by means of the olivary fasci-culi which decussate above the aqueduct of Sylvius and enter into minute relation with those ganglia) seem to be more directly connected with the lateral columns of the spinal cord. By means of the *processus a cerebello ad testes* and the Valve of Vieussens, they are brought into close relation with the cerebellum. In the lower orders of vertebrates— fishes, for instance—the cerebellum and optic lobes are more

integrated with each other than in the higher vertebrates.
That which apparently corresponds to the Valve of Vieussens
assumes much larger proportions, and enters more intimately
into the composition of the optic lobes than is apparent in
the homologous corpora quadrigemina of mammals.   Hence
the connection of the optic tracts with the cerebellum is of a
much closer nature than their mere outward distribution on
the surface of the optic lobes would signify.   When we con-
sider also the very close connection which subsists between
the superior crura of the cerebellum and the nuclei of the
third and fourth nerves, the ocular relations of the cere-
bellum become more and more evident.   But apart from
anatomical considerations, faradisation demonstrates the
oculo-motorial signification of the cerebellum.   What re-
lation it may have with the visual optic centres is not yet so
clearly ascertained.[1]

It is an interesting fact in connection with tumour of the
middle lobe of cerebellum that there is a distant tendency in
the recumbent posture to opisthotonus, as seen on the ex-
tension of the legs and the throwing back of the head. This
I am inclined to attribute to some degree of irritation of
the corpora quadrigemina, owing to the situation of the
tumour.

Though the cerebellum co-ordinates the eyeballs, the pro-
bability is that the voluntary alterations of the optic axes
are conditioned by the conjoint action of centres situated in
the cerebral hemispheres.[2]

The maintenance of the equilibrium of the body is inti-
mately bound up with the integrity of the oculo-motorial
centres.   The reeling gait, characteristic of tumours in the
cerebellum, and the impossibility of maintaining the equi-
librium in animals whose cerebellum has been removed, or

[1] The following quotation from Vulpian may be read with interest in connection
with this : ' Au contraire, les nerfs optiques n'ont assurément aucune relation
direct avec lo cervelet ; et pourtant, dans un grand nombre de cas de lésions de
cet organe, la vue s'est trouvée soit affaiblie, soit même abolie. Comment expliquer
ce désaccord entre les données anatomiques et les faits pathologiques ? Question
très-difficile à résoudre !'—*Leçons sur la Physiologie du Système nerveux,*
Paris, 1866, p. 615.

[2] See notes, p. 49.

only superficially destroyed, may not be regarded as depending on interference with the oculo-motorial centres alone. But other facts show that these are at least of essential importance. Most men have difficulty in co-ordinating their steps with their eyes shut. And in cases of locomotor ataxy, where the cerebellum is cut off from its normal relation with the spinal cord by disease of the posterior columns, it is utterly impossible for the patient to maintain his equilibrium with his eyes shut. The giddiness which accompanies nystagmus also points to the same importance of the ocular centres in the maintenance of the equilibrium.

Whether the cerebellum influences the co-ordination of the muscular movements concerned in the maintenance of the equilibrium in a voluntary manner, or whether it does so rather as a reflex or automatic balancing organ, through the combined action of the superior, middle, and inferior crura, and their various connections, is not yet certainly made out. That it acts consentaneously with the cerebral hemispheres is evident, but in what manner, and through which tracts, will require further investigation.

In regard to the movements of the eyeballs, it is to be remembered that each half of the cerebellum co-ordinates both eyes, and hence extensive disease of one half, such as abscess, may not apparently produce any marked symptoms so far as these movements are concerned.

The pathology of epileptiform convulsions, chorea, and epileptic hemiplegia, receive much light from the foregoing experiments. I regard them as an experimental confirmation of the views expressed by Dr. Hughlings Jackson. They are, as it were, an artificial reproduction of the clinical experiments performed by disease, and the conclusions which Dr. Jackson has arrived at from his observations of disease are in all essential particulars confirmed by the above experiments.

From a study more particularly of localised and unilateral epileptiform convulsions, and of the phenomena of hemichorea, and the relation of both these to certain forms of hemiplegia, combined with the results of *post-mortem* examination, Dr. Hughlings Jackson arrives at the conclusion

that the different epilepsies are due to ' discharging lesions,' and chorea to instability, of the cortical centres in the region of the corpus striatum.

To use his own words,[1] ' in a case of convulsion beginning *unilaterally*, I should believe that abnormal changes *did exist* in the region of the corpus striatum, though minute search had failed to *discover* them. A moment's consideration shows that such an inference is not an unreasonable one. For the alterations in grey matter, on which a convulsion or any " mobile " symptom (chorea, tetanus, &c.) directly depends, must be very slight; they are such that *function can still continue*, although it is disorderly function—it is a departure from health in the degree of exaggeration. The discharge in the convulsion is sudden, abrupt, and excessive, instead of being, as in health, slight, the result of *definite provocation and orderly*—but discharge there is.' In a footnote to this latter sentence he adds, ' It is assumed throughout that the cause of chronic convulsions (epileptic and epileptiform seizures) is a permanent condition ; there is grey matter, which, by nutrition, reaches abnormally high tension, and then discharges, on very general or very slight provocation. After the discharge the exploded grey matter again reaches abnormally high tension, and is again ready for explosion.'

A reference to the experiments specially made on rabbits and cats, for the purpose of inducing epilepsy as well as to conditions under which epileptic convulsions occurred, during the course of the subsequent localisation experiments, will show how closely this hypothesis corresponds to actual demonstrated facts.   While moderate stimulation of a centre caused merely the apparently normal excitation of the muscles co-ordinated there, more powerful stimulation excited an epileptiform condition of the same muscles, while diffused irritation of the whole hemisphere, by causing the current to travel from one end to the other, was sufficient to produce general convulsions, usually, but not always, restricted to the opposite side. In these general convulsions the animal apparently lost consciousness.

[1] ' Med. Times and Gazette,' May 10, 1873.

An abnormally irritable condition of the cerebral centres thus artificially excited was sufficient to cause repeated explosive discharges, on very slight provocation. This abnormal condition, once induced, caused such an epileptic habit that frequently localised exploration of the individual motor centres had to be given up on account of the readiness with which fits occurred. I have remarked it as a curious fact that in the rabbit it apparently took some seconds to work up the centres into their explosive condition, when the current was directed for some time through the whole hemisphere. The similarity between the conditions induced by faradisation, and those caused by the presence of a foreign body pressing on some part of the hemispheres, is manifest. The irritating effect of such a lesion is well characterised by Hughlings Jackson as a 'discharging lesion.' It does not destroy the cerebral centres, but by its presence keeps up in them an abnormally irritable condition, ready to discharge itself in epileptic convulsions with or without further provocation. It is instructive also for the purposes of diagnosis and exact localisation of the seat of lesion to pay particular attention to the march of the spasms, and the order in which the convulsions begin and terminate. Much attention has been paid to this subject by Hughlings Jackson, and remarkable harmony subsists between his clinical observations and the facts of the above experiments.

It is not unusual to find cases where unilateral epilepsy commences in one muscle, such as the zygomaticus, or in the muscles of the thumb and index finger, or it may be in the great toe.

Frequently, the motor spasm may remain limited, say to the fingers, but more frequently it becomes more general, the whole arm becoming convulsed. At this point, also, it may cease, or it may next invade the face, and afterwards become general. With the general convulsions of one side consciousness becomes lost, and the other side may likewise be invaded by the clonic spasms. The above experiments, in which I have described the march of the spasms in the cat and rabbit, and also the cases where epileptiform convulsions occurred during the course of the other experi-

ments, reveal similar phenomena, and also throw much light on their causation. On prolonged irritation of an individual centre, the muscles there co-ordinated became affected with clonic spasms, which in some cases remained localised; or, as frequently happened, the convulsions invaded other groups of muscles, selecting usually those whose centres had previously been irritated (I would call particular attention to the facts recorded in page 50). At other times the whole of the hemispherical centres had become so irritable that they discharged themselves simultaneously, even on very localised additional irritation. Yet, even when simultaneous diffused irritation of the whole of the centres was induced by causing the current to traverse the hemisphere from end to end, an order was observable in the course of the spasms. Experiments II. and III. illustrate this in a very striking manner. In the rabbit the convulsions usually began in the mouth and lips, and in the cat the eyelids and face first, next the shoulder and paw, and lastly the hind leg and tail were thrown successively into convulsions. Here we observe the muscles most in voluntary action are the first to be attacked. The centres for these are situated in the more anterior parts of the brain, and many facts have been given to indicate that these are more excitable and more easily discharged than those situated more posteriorly, such as the hind leg. Hence a general irritation of the whole hemisphere manifests itself primarily in the more excitable parts, and these coincide with the most voluntary centres. But, on the other hand, when the electrical current was made to traverse other centres with a greater degree of limitation (see Obs. 2, Exp. III.), the convulsions began in the muscles whose centres were there represented, and then spread to the more irritable anterior centres for the eyelids and face. The bearing of these facts on the diagnosis of the exact seat of discharging lesions in the hemispheres, such as tumours, is evident. When epilepsy begins in the hand, and particularly if it frequently occurs in the same manner, and more especially if it sometimes remains localised in the hand and arm, we have every reason for diagnosing a discharging lesion of some part of the superior frontal convo-

lution of the opposite hemisphere.[1] If, in like manner, it
has a tendency to begin and localise itself in the leg, the
probability is that the lesion is in the homologue in man, of
the leg centre of the superior external convolution in cats and
dogs. A tendency for the spasms to localise themselves in
the eyelids and face would, in like manner, in my opinion,
point to some lesion of the middle frontal convolution or its
homologue in man. If, on the other hand, it affects pri-
marily the mouth and tongue, leading to disorders of articu-
lation, it is in one or other lower frontal convolution, in the
region of the fissure of Sylvius.[2] The simultaneous discharge
of all the centres will of course indicate nothing as to
seat of the lesion, for it need not depend on localised
irritation.

In view of the facts demonstrated by the foregoing series
of experiments, I do not deem it necessary, in stating what
on the basis of these facts I consider to be the essential nature
of epilepsy, to enter into any lengthened examination of the
various theories which are entertained as to the pathology of
this disease. My conclusions are entirely in harmony with
those of Dr. Hughlings Jackson, who regards epileptic con-
vulsions as essentially dependent on ' discharging lesions ' of
the cortical centres. Many of the difficulties in the causation
and definition of the epileptic state will disappear, if we regard
the phenomena of this disease, or condition, as proximately de-

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[1] This looks like *post factum* prediction, for a similar diagnosis grounded on clinical
facts by Hughlings Jackson has been triumphantly verified. I extract the following
sentences from an article by him in the ' British Medical Journal,' May 10, 1873.
' In the " Medical Mirror " of September, 1869, I published the case of a man who
had fits affecting the *right* arm. In this case there was a tumour in the hinder
part of the first (uppermost) frontal convolution of the left hemisphere.' ' Lately,
I was allowed by Mr. Soutter to see a patient of his, who had literally innumerable
fits limited to the right arm. Mr. Soutter witnessed many ; I saw several.
Shortly before the patient's death she had, Mr. Soutter tells me, universal con-
vulsion. *Here I correctly predicted disease of the hinder part of the first (upper-
most) frontal convolution.*' (The italics are ours).

Allowing for greater anterior development of the brain in man, the correspond-
ence of this region with the regions for the fore paw in cats and dogs is signifi-
cantly indicated, and confirms in a most gratifying manner the results and con-
clusion of my own experiments.

[2] I have now (June 14) ascertained the position of all these centres in the brain
of the monkey, and therefore, by implication, their situation in man. These ex-
periments will soon be published.

pendent on a local, or general, abnormal condition of the cere-
bral hemispheres, characterised by the tendency to recurrent
sudden and explosive discharge of the motor centres, with or
without functional perversion of the centres which manifest
themselves inwardly in consciousness.   We shall thus be
enabled to recognise in their true light, and place in their
proper relations to the more generally recognised type of
epileptic seizure, those forms of epilepsy which commence
unilaterally in one or more groups of muscles, (and which
generally indicate some localised gross lesion of the cerebral
hemispheres,) while at the same time we shall afford a satis-
factory explanation of the phenomena of idiopathic epilepsy,
so called, because it has not as yet been shown to be depen-
dent on any constant discoverable lesion.   For, as we have
seen, the local irritation which manifests itself in a limited con-
vulsion has a tendency to become diffused, and to invade the
whole of the cortical centres, so that what at first was merely
a local convulsive spasm without affection of consciousness,
may gradually gain in range and intensity, until, along with
the motor perversion we get the loss of consciousness, which
is regarded as an essential factor in the true epileptic seizure.
In these local and unilateral attacks we have a successional
analysis of the phenomena which a so-called idiopathic attack
presents in too complicated a form for successful diagnosis as
to essential nature and causation.   But in such cases also
there are numerous facts which indicate that the seat of the
motor and psychical disturbances of the epileptic attack is
above the medulla oblongata, in centres which probably have
an intimate local relation with each other.   And now that the
motor signification of the grey matter of the hemispheres is
clearly demonstrated, it is not necessary to assume that the
medulla oblongata is the primary seat of the motor disturb-
ances, while the psychical symptoms are only subordinate to
changes induced in the circulation of the brain by a primary
affection of the medulla itself.   When it is said that the
proximate cause of the epileptic seizure is a condition of insta-
bility of the cortical centres, nothing is implied as to the
exact physical condition which may exist.   It may be esta-
blished as a hereditary tendency, and it is, as we know, capable

of being induced by numerous methods of centric or excentric irritation both in the indirect experiments of injury and disease, and as the results of direct experiment, such as blows on the head (Westphal), or injuries to nerve trunks or the spinal cord (Brown Sequard).

It is quite possible that convulsions such as are called epileptiform convulsions may be induced by irritation of motor tracts beneath the cortical centres, and without the intervention of the latter, or even in their entire absence, as in the recent experiments of Brown Sequard. But such convulsions as are caused by direct irritation of the medullary centres, or indirectly by conditions of blood or otherwise, which act primarily on the vital centres of respiration and circulation, as well as those which are more clearly traceable to spinal reflex origin from temporary causes, appear to be capable of distinct separation from the class of cases usually recognised as truly epileptic, in which a habit is established, and in which the psychical and motor disturbances are evidently of primary central or cerebral origin; while the disturbances of the respiration and circulation, such as exist, are to a great extent only the secondary result of the convulsive and irregular muscular action. All the other phenomena of a universal epileptic seizure, such as dilatation of the pupils and distortions of the optic axes, may be accounted for by diffused irritation and explosive action of the corpora quadrigemina and cerebellar oculo-motorial centres along with the cortical centres themselves. The psychical perversions may be supposed to depend on the same conditions of nerve tissue as lead to explosive muscular action, or at least that they stand in causal relation to each other. And it will form a valuable subject of future research, though not to be answered by experiments on the lower animals alone, to determine whether the disorders of perception and volition of the 'petit mal' are, as is probable, only abortive stages in the development of the 'grand mal,' and affections of centres and nerve elements in close geographical relation to those which are subservient to motor acts of volition; or whether those forms of sensory epilepsy, characterised more especially by temporary (often followed by permanent) symptoms of mental

alienation, referable primarily to disorders of sensation and emotion, are dependent rather on the same abnormal condition of only the posterior parts of the brain, which are more probably connected with these mental processes.   Disorders of sensation, emotion, volition and intellect, temporarily or permanently accompanying or following epileptic attacks, receive their explanation in the general affection of the whole brain in the fully developed grand mal, but it is to clinical observation and minute pathological inquiry in special forms, presenting individual peculiarities, that we must look for more definite localisation in the cerebral convolutions of the centres for mental processes not in immediate relation to motor acts.

The movements which are seen in chorea bear an intimate relation to those of epilepsy, and indicate the same centric causation. 'They are not mere spasms and cramps, but an aimless profusion of movements of considerable complexity, much nearer the purposive movements of health.   They are not so much incoherences of muscles (like the " fist " we see in a partial fit of those convulsions which begin unilaterally, where all the muscles of the hand are in action at once), as incoherences of *movements of muscles*.   There is some method in their madness.   They are not analogous to playing at once many keys of a piano in mere order of continuity, but to a random playing of harmonious chords.   Again, they are *successions* of movements ; moreover, they are successions of *different* movements.' [1]   I regard the above experiments in this case also as an experimental demonstration of the accuracy of the views of Dr. Hughlings Jackson, who places the proximate cause of these movements in an unstable condition of the grey matter of the cerebral convolutions.   In fact, the localisation experiments may be looked upon as a method of geographically fixing the motor signification of the cerebral convolutions by the induction of an artificial chorea.   A momentary application of the electrodes to a cortical centre caused a choreic twitch of the muscle or group of muscles there represented.   On the other hand, a longer stimulation resulted in tonic spasm, while prolonged irritation resulted in

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[1] ' Observations on the Physiology and Pathology of Hemi-Chorea,' by Dr. Hughlings Jackson, ' Edin. Med. Journ.,' 1868, p. 297.

clonic spasms of the same muscles, just as if the centre were repeating its tonic stimulation. And still further repetition of the stimulation of the same centre frequently resulted in diffused irritation of all the centres at once, as manifested in a genuine epileptic attack.

The relation of choreic twitches to epileptic convulsions is thus rendered tolerably manifest. The difference is essentially one of degree and order rather than of kind. While the instability of the cortical centres in chorea is characterised by frequently repeated momentary discharges in random succession, that of epilepsy is a periodic, abrupt, and more or less simultaneous perversion and explosion of all the centres.

In chorea it would seem as if the motor centres had in some manner become detached from those which guide and direct them ; as if they had a will of their own, and delighted in their freedom. They resemble a horse which has got the bit between its teeth and broken loose from the control and direction of its rider, whose excitement and efforts to recover his command act only as so many stimuli to redoubled action on the part of the runaway steed.

What the physiological or anatomical state of the nerve-cells may be on which both epilepsy and chorea seem to depend, and whether the vascular and tissue changes frequently seen in the brain and membranes of epileptics are the result, or the cause of the abnormal condition of the cells themselves, are questions which must be left for minute pathological investigation to answer.

From the functions of the cerebellum as a co-ordinating centre for the muscles of the eyeballs, and what has been said on the subjects of epilepsy and chorea, it is not difficult to trace nystagmus to a similar condition of the cerebellar centres. It would be not unjustifiable to define the cause of nystagmus as a choreic or epileptic condition of the cerebellar oculo-motorial centres, concerned in the special movements of the eyeballs which are affected. What these are in the human cerebellum will have to be answered by comparative anatomy and further physiological experiments. The rolling of the eyeballs and distortion of the optic axes which form part of the fully developed epileptic seizure, are evi-

dently the extension of the affection of the cerebral centres to those of the cerebellum.

The conditions which lead to the state of the centres on which nystagmus depends, whether of centric or eccentric origin, or both, will have to receive further light from the investigations of ophthalmologists, and the relations of diseases of the cerebellum to diseases of the eye must form important subjects for future research.

Imperfect though the foregoing paper may be in many details, and requiring much additional investigation to render it complete, the way has been opened, and a new light has been thrown on many obscure points in cerebral physiology and pathology. Though many of my conclusions are in opposition to commonly received views, I think that they are not altogether devoid of some tangible foundation on novel facts of experiment.

The summary of the more important results and conclusions which I published in the ' British Medical Journal ' for April 26, 1873, I again reprint with few alterations [in brackets] as an abstract of the foregoing :

1. The anterior portions of the cerebral hemispheres are the chief centres of voluntary motion and the active outward manifestation of intelligence.

2. The individual convolutions are separate and distinct centres ; and in certain definite groups of convolutions (to some extent indicated by the researches of Fritsch and Hitzig) and in corresponding regions of non-convoluted brains, are localised the centres for the various movements of the eyelids, the face, the mouth [and tongue], the ear, the neck, the hand, foot, and tail. Striking differences corresponding with the habits of the animal are to be found in the differentiation of the centres. Thus the centres for the tail in dogs, the paw in cats, and the lips and mouth in rabbits, are highly differentiated and pronounced.

3. The action of the hemisphere is in general crossed ; but certain movements of the mouth, tongue, and neck are bilaterally co-ordinated from each cerebral hemisphere.

4. The proximate causes of the different epilepsies are,

as Dr. Hughlings Jackson supposes, 'discharging lesions of the different centres in the cerebral hemispheres. The affection may be limited artificially to one muscle, or group of muscles, or may be made to involve all the muscles represented in the cerebral hemispheres, with foaming at the mouth, biting of the tongue, and loss of consciousness. When induced artificially in animals, the affection as a rule first invades the muscles most in voluntary use, in striking harmony with the clinical observations of Dr. Hughlings Jackson.

5. Chorea is of the same nature as epilepsy, dependent on momentary [and successive] discharging lesions of the individual cerebral centres. In this respect Dr. Hughlings Jackson's views are again experimentally confirmed.

6. The corpora striata have crossed action and are centres for the muscles of the opposite side of the body. Powerful irritation of one causes rigid pleurosthotonus, the flexors predominating over the extensors.

7. The optic thalamus, fornix, hippocampus major, and convolutions grouped around it, have no motor signification [and are probably connected with sensation].

8. The optic lobes or corpora quadrigemina, besides being concerned with vision and the movements of the iris, are centres for the extensor muscles of the head, trunk and legs. Irritation of these centres causes rigid opisthotonus [and trismus].

9. The cerebellum is the co-ordinating centre for the muscles of the eyeball. Each separate lobule (in rabbits) is a distinct centre for special alterations of the optic axes.

10. On the integrity of these centres depends the maintenance of the equilibrium of the body.

11. Nystagmus, or oscillation of the eyeballs, is an epileptiform affection of the cerebellar oculo-motorial centres.

12. These results explain many hitherto obscure symptoms of cerebral disease, and enable us to localise with greater certainty many forms of cerebral lesion.

In conclusion, I desire to express my warmest thanks for much valuable and indispensable assistance in observing and

recording the results of experiments to Dr. Crichton Browne, Dr. Milner Fothergill, Mr. J. C. Galton, Dr. McDowall, at Wakefield, and, in more recent experiments, to Dr. Lauder Brunton.   To Mr. Galton I am likewise indebted for the original sketches and subsequent drawings on the wood of the illustrations which accompany this paper.

OBSERVATIONS

ON THE

HISTOLOGY OF THE BRAIN

IN THE INSANE.

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In the last volume of these Reports I recorded the morbid appearances presented by the cortical substance of the brain in a typical case of Chronic Brain-Wasting, as contrasted with those conditions observed in the perfectly healthy organ. I pointed out that the general structure of the former was so different from that of the latter as to render the presence of an alteration, when two sections were placed side by side and examined under similar conditions, evident and conclusive. I showed that, of the three great elements of which the cortical substance is composed, viz., cells, vessels, and intercellular substance, the first were decidedly abnormal in their number, characters, and distribution; the second participated in the change, presenting appearances not seen in health, and the third presented little alteration.

But from one such examination, however carefully conducted, no reliable conclusion could be drawn; and in recording the case, I offered none, leaving that to be elicited by further examination of similar cases and comparison with others.

An opportunity for further investigation has been furnished me by the case of N. N., male, æt. 44, who was admitted

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into this asylum, December 29th, 1871. He then presented very distinctly those symptoms and general characteristics which are observed in cases of chronic brain-wasting, and which I referred to in my last paper. He grew gradually but steadily worse after admission, and died January 23rd, 1873, when the condition of atrophy diagnosed during life was verified by *post-mortem* examination. The whole brain weighed 47 ozs., $3\frac{1}{2}$ ozs. of serous fluid escaping during its removal. The arachnoid was thickened and cloudy over the frontal and parietal lobes, the convolutions of which were considerably wasted and the sulci broad and filled with fluid. The pia-mater stripped very freely, and there was no atheroma of the vessels at the base.

It will be observed that the above case coincides closely with that of L. A., an examination of which formed the subject of my previous paper. In both there was a condition of atrophy coming on at a comparatively early period of life, characterised by similar symptoms, and after death by closely allied pathological conditions, viz., atrophy of the brain with compensatory effusion, and thickening of its membranes without adhesion.

But there is another morbid state of the brain which, in many instances, cannot, by the unaided vision, be distinguished from atrophy occurring in early or middle life. I refer to the state of senile atrophy. Are then the histological conditions presented by a case of chronic brain-wasting, the result of mental shock or overstrain, similar to those found in one whose end has been ' second childishness and mere oblivion?' In general paralysis, again, we have a disease, the characters of which clinically are, as a rule, well defined; but with regard to its morbid anatomy, notwithstanding all that has been written and said, I do but express my individual opinion when I say that the characters as usually described are not satisfactory. Granted that the experienced observer is able to fix on the nature of the case, from the general condition presented, with a considerable degree of probability, yet instances will occur in which this cannot be done. In such cases, the all-important question arises whether there is any histological condition which, by its

presence or absence, will settle the question. This question, as far as it is in my power to do so, I shall now consider with reference to such cases as I have examined, commencing with one of chronic brain-wasting.

Chronic Brain-wasting.—When prepared sections of healthy brain are placed under the microscope, by far the most conspicuous objects are the cells; but under many morbid conditions, and very decidedly so in the present instance, that is not the case, the vessels, by reason of their size and general appearances, being the most prominent objects seen. The delicacy of the vascular structures so constantly found in health exists no longer, and in its place are found conditions which even the unpractised observer would at once recognise as being widely different. With regard to the smallest capillaries, however, I must confess I have not been able to make out any decided change in their structure, even when isolated, and under circumstances therefore most favourable for the display of anything morbid. But setting these aside, all the vessels in every part of the cortical substance in which I have examined them are decidedly diseased. I find that both arteries and veins are larger than in health. Now, the size of a vessel when viewed under the miscroscope may depend on increased thickening of the vascular wall by thickening of its coats or by morbid deposits, or, again, on dilatation and distension with blood. In the present instance both conditions are found to exist, the former being most prominent in the arteries, the latter in the veins. With regard to the changes in the walls of the vessels, giving to them the increased coarseness to which I have referred, the most important here found is the enormous multiplication of the nuclei. In health these bodies are few in number, oval in form, and not deeply stained by carmine; but here, in addition to these, the whole vessel is seen to be crowded with small nuclear bodies, closely but irregularly scattered over the vessel. They are exceedingly minute, round in form, and deeply stained—quite different, therefore, from the larger oblong nuclei which normally exist. That these minute corpuscles exist in health is undoubted, but in nothing like the number here discovered.

I would here observe, as a point I think worthy of notice, that it is in the deep, rather than in the superficial part of the cortical layer, that the disorganisation of the vessels is most distinct. I have frequently observed a small vessel in which, at the commencement of its passage downwards I could detect little or no alteration from the normal condition, soon alter its characters, and assume, more and more, conditions undoubtedly morbid.

But in addition to this aggregation of nuclei, other deposits on the vascular walls are found to exist, and of these the most distinct are masses of hæmatoidin and fatty granules. Now, a vessel in which the degenerative process is farthest advanced, presents all these conditions, its proper structure being almost completely masked. In a vessel seen in transverse section, these several points in structure can be well appreciated. In the centre the wide lumen of the vessel, in many instances filled with blood; around this the wall thickened and altered by proliferation of nuclei and morbid deposit; and surrounding this again, in some instances closely, in others at a considerable distance, the circular fibres of the cerebral stroma. With regard to the so-called peri-vascular canals, I must confess to some uncertainty. Here, however, the size of these canals is usually greater than can be accounted for by any shrinking of the vessels; but, on the other hand, round certain vessels no canal can be found intervening between them and the cerebral substance. On the whole, I am of opinion that peri-vascular canals, and these of considerable size, are among the conditions here presented. It is much to be regretted that by none of the ordinary means at present at our disposal can this point be studied while the brain is in a fresh state, and before hardening re-agents have introduced a source of fallacy. Were it otherwise, this point would be set at rest for ever.

Little need be said as to the course which the vessels pursue. In a few instances, I have observed some tortuosity, but not such as I would venture to designate morbid. In no case have I been able to demonstrate either kink or varicosity.

The hyaline sheath is not very distinctly seen, although

evidence of its presence is not wanting. In all cases I have found it to be perfectly hyaline, and without the tendency to fibrillation described by some authors.

In describing the cells and their method of arrangement, it is convenient to consider all those bodies which partake of the characters of cells or nuclei, and their mode of distribution in the cortical layer, from without inwards; that is to say, first considering the external layer, and gradually working downwards towards the central white substance of the convolution.

In pursuance of this plan, the *external layer* first demands attention. Here I find a waut of that delicacy and homogeneity which it presents in health. This, I think, is chiefly due to an increase in the number of the minute neuroglia corpuscles. But in addition to these, I have observed other bodies having very different characters. In size they vary considerably, but average between $\frac{1}{3000}$th and $\frac{1}{4000}$th of an inch in their longest diameter; they are but slightly tinted by carmine, very irregular in their outline, and without careful observation may easily escape notice. Their chief peculiarity, however, consists in the large number of extremely delicate branches which they possess. I believe that these bodies have been considered morbid, and a result of transformation of the neuroglia. That they may be morbid in the external layer, though scarcely probable, is possible, for I have as yet failed to demonstrate their existence here, in my healthy specimens; but that they do exist in health is undoubted, for in all my preparations of a brain, the healthiuess of which cannot admit of doubt, they are to be found both in the deeper layers of the grey matter and in the white. In most instances they contain a distinct nucleus, and when seen, as they sometimes occur, close to each other, their irregular bodies and long delicate branches intertwining and joining, present an appearance which is very peculiar. As to the nature of these bodies, I can form no decided opinion, but the important fact at preseut is that they are not morbid. It is important also to know, as I before remarked, that owing to their extreme delicacy, added to the fact that they are only very slightly affected by carmine, careful focusing

with a moderately high power is requisite to bring them
into view.   The increased coarseness of the external layer
manifests itself more especially at its junction with the one
beneath it. Under normal circumstances, the line of sepa-
ration is very distinct, but here it is not so, the one merging
more gradually into the other.

The *second cortical layer,* as in health, forms a broad
band of cells, somewhat ill-defined on its deeper aspect, and
also, as in health, is made up of three varieties of corpuscles.
1st. Small oval or pyramidal nerve cells.   2nd. Round or
oval bodies, having an average size of about $\frac{1}{4000}$th of an
inch, a delicate granular appearance, and a minute nucleus ;
they are but slightly stained by carmine.   3rd. Small nuclei,
having a homogeneous appearance, without central spot or
nucleolus, and deeply stained (neuroglia corpuscles).

With regard to the first class I have mentioned, viz. the
oval and pyramidal nerve cells, I cannot detect any deviation
from the average number of health, but in other respects
they are abnormal.   In health even these smaller cells are
characterised by a distinctness of outline, and in prepared
sections, by the deeply stained nucleus.   Here, in most
instances, both these qualities are wanting.   And this leads
me to remark, with regard to the staining of carmine, what
a valuable indication it forms in many instances, as to
the health and activity of a tissue.   True, with a sufficiently
strong solution and prolonged immersion, any tissue may be
deeply stained ; but, *cæteris paribus,* I think it will be found,
as in the present case, that the morbid specimens take the
carmine less readily than the healthy.   In health, we have
active, well-formed, deeply-stained cells ; in disease, and
notably so in the present case, small cells, ill-defined, and
but slightly stained.

No change in the structure of the two other forms of
corpuscles which go to form this layer can be detected, but
their number is decidedly increased.   In my former paper I
remarked that there appeared to be no deficiency in the
number of cell elements taken collectively, and further obser-
vation has fully shown this to be the case ; there is, on the
contrary, an excess. This excess must, I think, be attributed

to these smaller bodies, and more especially to the minute neuroglia corpuscles. Not only in this layer, but throughout the whole tissue, these bodies are thickly scattered, and are more especially numerous on and in the neighbourhood of the vessels, in the morbid appearances of which they play, as I have before described, an important part.

The layer above described passes gradually into the succeeding one, in which the largest nerve cells are situated. The separation of this layer from those adjacent is much less definite than in health, owing, as I have before shown, to deficiency in the number of cells. With regard to the structure of the individual cells, some do occur in which, with the highest powers at my disposal, I have failed to detect anything abnormal; but this is exceptional. In most instances, although the average size of the cells is not altered, they exhibit here, as in the previous layer, a want of sharpness and distinctness of outline, their continuations are torn, and few in number, and the cell itself is rounded and deformed. The nucleus partakes in the general change. This body, as seen in the large pyramidal healthy cell, follows more or less closely the form of the cell in which it lies, and is deeply stained. Here, on the contrary, with very few exceptions, it is round or oval and little stained. But although altered in form, it is invariably of large size, sometimes completely filling the enclosing cell, or even causing a projection from it. I believe also that I have been able to detect cells destitute of cell wall, and surrounded only by a little molecular matter, so that my belief in the theory, which I think I am right in attributing to Dr. Tigges, has been strengthened, and I hold with him that many of the corpuscles are actually nuclei which have been set free by the degeneration of the enclosing wall. The difficulty lies in the fact, that bodies having characters hardly to be distinguished from nuclei are to be found in other parts of the cortex and in situations far removed from the large pyramidal cells whose nuclei they resemble.

The next layer is more defined than any of the others. It is made up chiefly of small oval nucleated cells, and does not call for special notice. More deeply, the cells and nuclei

become very abundant, and present a confused mass, in which no definite strata can be distinguished. It is here more especially that the excess of the neuroglia corpuscles is most evident, not only under the microscope but to the naked eye, when a healthy and morbid specimen are examined together. In the former the external grey matter is deeply stained and sharply defined, in the latter it is much less so, owing, as I think, to the excess of these bodies which are so deeply stained by the colouring matter.

As to morbid deposits, the most common are hæmatoidin, colloid bodies, and fatty granules. The first, as before stated, is scattered abundantly over the vessels. Colloid bodies I have found on the surface of the convolutions, as well as more deeply, and also as a deposit, on the vascular walls. Fat granules are rare. I have in addition observed bodies which I find have been already noticed by Dr. J. Batty Tuke, and well compared by him to the spores of the favus fungus. In my experience they are usually found in the neighbourhood of the vessels. They are homogeneous, of a pale yellow colour, highly refractive, and unaffected by carmine. Of their nature we are at present ignorant.

I have thus described generally the appearances I have found in this case. But though in all parts I have been able to detect similar changes, it is right I should add, not to the same extent. I believe them to be most marked in the frontal lobe, and to diminish from before backwards, being therefore least seen in the occipital region.

In summing up the histological conditions present, it will be observed, that they closely agree with those I described in my last paper. With respect to the cells :—

1st. Increase in their number taken collectively, this increase being due to an excess of the small corpuscles.

2nd. Deficiency in the number of the large pyramidal cells, a want of distinctness in their outline and branches, the nuclei of large size, but altered in form, and but slightly stained by carmine.

3rd. Increase in the density of the outer nerve layer, and want of definition in the others, as compared with healthy tissue.

With respect to the vessels :—

1st. Great increase in their size, due in most instances to a condition of dilatation, with thickening of the walls by proliferation of nuclei and morbid deposits of hæmatoidin, fat, &c.

2nd. The presence of large perivascular canals.

It will be noticed that I do not include in the above any alteration in the course of the vessels. In this respect I have failed to discover anything abnormal, or at all approaching the kinks or knots which have been described in some other forms of cerebral disease.

Senile Atrophy.—In comparison with the above case, I shall now proceed to describe the histological appearances presented by a typical case of senile atrophy. It was that of a woman, B. M., who died in this asylum November 6, 1872. The *post-mortem* condition of the brain was shortly as follows :—

The whole brain weighed 40 oz., 6 oz. of serous fluid escaping during its removal. The arachnoid was thickened and cloudy over the whole of the frontal and parietal lobes, but not over the occipital lobes or at the base. The pia-mater stripped very freely. The grey matter was pale and thin, the medullary substance of a dirty appearance, the puncta-vasculosa being unusually numerous. The ventricles were of large size, filled with clear fluid, and the vessels at the base were atheromatous.

The condition of the vessels of the cortical layer in this case forms a marked contrast with that of those before described. Under a low power, indeed, they would in most cases be pronounced quite normal, and it is only when higher powers are brought to bear, that appearances not seen in health are found to be present. The most prominent and indeed the only one of importance, seems to be the deposit of fatty granules and small compound granular bodies on the walls of the vessels. The latter are not numerous, and are usually met with lying here and there in the hyaline sheath. With regard to the fatty granules, they are met with either scattered in an irregular manner over the vessel, or more commonly collected together. In the latter case they may be seen forming a projection from the vascular wall, or filling

up the angle formed by diverging branches. The hyaline sheath of the vessels can be traced with unusual distinctness, serving as it does for the deposit of fat and other particles, which assist in giving it prominence.

Fatty and pigmentary degeneration of the cells are the most important and prominent morbid conditions. The degeneration is most observable in the large pyramidal cells, in which, in its earlier stages, it gives rise to a slight bulging of the cell, the nucleus, however, preserving its normal position and characters. In more advanced stages the bulging and deformity is found to be greater, the branches are no longer visible, and the nucleus is atrophied and pushed out of position. In the last stages the whole cell is reduced to a simple mass of granules, no branches, cell wall, or nucleus remaining. It must not be supposed that in the case of a few cells only is the degeneration I have described to be observed; on the contrary, it is rarely that a cell having fairly normal characters is to be seen, and even then signs of commencing atrophy are almost always present.

The small neuroglia corpuscles, by their number, constitute another distinct point of difference between the present and the previous case. Here, if anything, they are below the average. On the other hand, the larger granular bodies formerly described would seem to be more numerous, for the number of cells of all kinds seen at any time in the field of the microscope is certainly not below the average.

In several instances patches of degeneration have been observed in the grey matter, the proper nervous elements being broken down and converted into molecular *debris*. The structure of the neuroglia in all cases is loose and imperfect.

I am sorry I cannot point to the condition I have above described as being invariably present in cases of senile atrophy. The case of J. C., which I shall next allude to, negatives this conclusion. This patient died at the age of 69 years, his brain after death presenting all those characters so well known in connexion with old age :—the wasted, waterlogged convolutions; the thickened membranes, and atheromatous vessels. It is right, however, I should add that his symptoms during life differed in some important respects from

those of B. M., being those of maniacal excitement, super-
added to the dementia. In him also death occurred after an
illness of only slightly more than a year; whereas iu the
former case, the patient for years was utterly demented, main-
taining merely a vegetative existence.

In the present case, the vessels of the cortical substance ex-
hibit conditions which I am unable to distinguish from those
I formerly described at length in connexion with chronic
brain wasting, and which contrasted so forcibly with those
observed in the last case of senile atrophy. I can offer no
explanation of this discrepancy between analogous cases, for
I cannot conceive it possible that the fatty degeneration, as
before described, can be a further stage in the condition here
presented. I must be content therefore to record the fact,
and leave the explanation to the future.

The cells also present notable points of difference, for in
the case of B. M. a condition of fatty degeneration was
almost invariable, while here it is rare. They are decidedly
numerically deficient, and of small size, as if shrunken. The
body of the cell displays in most instances a remarkable ab-
sence of colour, being unaffected by carmine, and the natural
pigment granules almost absent. On the other hand the
nuclei are very distinct, being well formed and deeply stained.
I have occasionally observed a cell represeuted by nothing
more than a mass of granules, but this, as I before stated, is
quite exceptional. Of the other corpuscles those of the neu-
roglia are by far the more numerous, and in this again the
present contrasts strongly with the previous case. As to
whether in both the same degeneration of the large nerve
cells exists, the appearances being modified by the stage of
the process, I would guard myself against the expression of
any positive opinion; but at present I do not think that such
can be the case.

General Paralysis.—In four cases of general paralysis I
have studied minutely the appearances presented by the grey
matter of the hemispheres, for purposes of comparison with
each other, and with the cases before described. The first I
shall consider is that of J. M., who died in the last stage of the
disease, and whose brain after death presented, in the dense

adhesion of the pia-mater, the most common and significant pathological condition.

A dilated condition of the vessels seems to be the change most commonly present. It will be remembered that the same remark was made with respect to the vessels in chronic brain wasting, and indeed in many particulars I must confess that the resemblance is very close. Here, however, as a rule, the coats of the vessel are not thickened, and the proliferation of nuclei is not so marked. On the other hand the hyaline sheath is more distinct, and is the seat of abundant deposits of hæmatoidin all over the vessels. With regard to the course which the vessels pursue, in some instances I have observed unusual tortuosity, but not to such an extent as to produce a kink. In no case has anything approaching fatty degeneration of the walls, as described by some observers,[1] been detected. It will be observed then that I am unable to draw any accurate distinction between the vessels in the present case and those in brain wasting.

The layers of the grey matter are very distinct. The outer layer is not of abnormal density, contains an average number of small neuroglia corpuscles, and is well defined from the layer which succeeds it—it presents in short the appearances of health. With regard to the individual nerve cells, I can detect nothing abnormal in the smaller varieties, and even the large pyramidal cells, in most cases, present characters to all appearance perfectly normal; they are well formed, sharply defined, their branches are distinct, and their nuclei deeply stained. Two conditions, however, may be observed, both decidedly pathological, and therefore important. I refer in the first place to a loading of the cell by pigment granules. This appearance, as seen in general paralysis, was, I believe, first described by Dr. Lockhart Clarke.[2] A cell thus affected loses almost entirely its normal characters; though as a rule but little altered in size, it is, as it were, rounded and inflated, and its branches having disappeared and the nucleus gone, there remains nothing but a mass of

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[1] ' Recherchos sur l'Anatomic pathologique et la nature de la Paralysie générale, par les docteurs Poincaré et Bonnet.'

[2] 'Lancet,' September 1, 1866, p. 229.

pigment granules loosely held together. I must repeat, however, that this transformation is quite exceptional. The other condition to which I have referred is a most peculiar one, and such as I have never before met with either in health or disease. It consists in the presence of nerve cells of immense size, situate about midway in the depth of the cortical layer. Standing out like giants among the other cells, they present an appearance which cannot fail instantly to attract attention. In shape they vary considerably, the majority approaching more or less the pyramidal form, but often being very irregular in their contour. Their branches are both large and numerous. I have counted as many as eight or ten proceeding from a single cell. The nucleus is large but not proportional to the size of the containing body, the density of which masks its contents. In number, these large cells, when compared with the others, are relatively few; usually not more than a dozen or so being seen in one specimen, but sometimes fewer, and occasionally none at all. I find them most numerous in the parietal region, then in the occipital, and rarest in the frontal lobes. Now these bodies present an appearance altogether different from those last described; cell wall, nucleus, and branches being all very distinct, while, as regards size, they have no rivals. As to their pathological significance, I am unable at present to form an opinion; the point is one for further enquiry.

There remains but little to be said with respect to the other cell elements. The number of the smallest neuroglia corpuscles is certainly not so great as in brain wasting, and this in the present case forms an important distinction. The peculiar small branching corpuscles formerly described are here also to be met with, but are not numerous. The fibres, so far as I have been able to observe them, are not destroyed, but present a tortuosity and irregularity in their course such as I have not found in the healthy brain.

The histological appearances found in the case of R. L., male, who also died in the last stage of general paralysis, differ in some important particulars from those observed in the former case, and to a consideration of these differences I shall now pass.

The vessels do not display the proliferation of nuclei on which I have before laid so much stress. Occasionally small masses of hæmatoidin are seen, but as a rule the vascular walls are free from morbid deposit. On the other hand I have frequently been able to observe the looped, tortuous, and occasionally also the varicose condition so frequently described in connexion with general paralysis. It must not be imagined, however, as I fear is too often done, that these conditions when existing are to be seen in all the vessels, or even in the greater number. As far as my experience goes, tortuosity is common enough, but the others are quite exceptional. Surrounding the vessels perivascular canals are usually to be seen ; they do not, however, present a very prominent feature in the present case.

An abnormal condition of the cells here, as in the previous cases, is distinctly observed. The body of the cell is usually shrunk, the wall being closely applied to the nucleus, which is of large size, more or less round, and but slightly stained. In the more advanced degenerative stages no cell wall is observed, the nucleus alone, or surrounded in part by molecular *débris,* being all that is left. There exists also, however, another condition which has been described by some as ' inflated.' The size of the cell is not much altered, but it exhibits an irregular inflated appearance, this being not due to excess of pigment or fatty degeneration. It would seem probable that this is the earlier stage, and is succeeded by the shrinking and atrophy before described. With regard to the various layers of the grey matter, the most external is here peculiar, presenting a delicacy and whiteness to a greater degree even than in health, and contrasting strongly with all my other morbid specimens.

The small branching corpuscles to which I have already often referred, are here found, and present their usual characters.

Patches of molecular degeneration are frequently observed in the white substance, the proper nervous elements being pushed aside and destroyed. These spots, which can be very readily seen with the naked eye (at least after the brain has been hardened, for I have not been able to observe them

in the fresh state), present all the characters described by Dr. Tuke and Professor Rutherford, and called by them ' miliary sclerosis.'

The next case of general paralysis is that of J. S., male, which ended fatally after a course of eighteen months' duration. The appearances presented by the vessels in this case correspond very closely with those described in that of J. M.; my remarks, therefore, need be but few. The prevailing condition, which is more especially observed in the veins, is one of dilatation. There is as a rule no thickening of the vascular walls and no great proliferation of nuclei, but in all cases abundant deposit of hæmatoidin. They appear to pursue a normal course, and in very few instances have I been able to observe perivascular canals, and these not of larger size than can be accounted for by the hardening process.

With very few exceptions all the nerve cells, but more especially those of larger size, are seen to take part in certain changes. But here again I need say very little, the conditions being, as far as I am able to judge, identical with those observed in the last case, viz., an inflated appearance of the cell wall, occurring as the first step in the process of atrophy; the nucleus, however, remaining large, rounded, and stained with difficulty.

The case of T. B., whose symptoms were in every respect most typical of this disease, forms the last I shall for the present refer to. The patient died after an illness of more than two years' duration, and in the last stage of the affection.

The vessels in this case present the same characters as I have described in connection with two of the previous cases of general paralysis and also in chronic brain wasting, viz., dilatation, with copious deposit of nuclei and pigment, but without any marked irregularity in their course.

A condition of simple atrophy of the cells is that most generally met with, the excess of pigment and inflated appearance before described being altogether absent. It is · worthy of remark also that the nuclei of the large cells

are not swollen and rounded, as was observed to be the case in the former instances of general paralysis.

It remains for me now to state what conclusion I would draw as the result of the observations I have recorded.   But if these have been closely followed, it will be seen that I am bound in candour to record failure—failure in this ; that while in every case I have been able to demonstrate the presence of important morbid conditions, I am not able to point to any, which, by their presence or absence, will, of the three forms of disease which I have considered, mark infallibly the nature of the affection.

I look forward with confidence to the time when it will be in our power to do this ; but, in the meanwhile, believing as I do that in faithful descriptions and careful comparison lies the road to the truth, I have ventured to record these few observations, as being perhaps not altogether destitute of interest or importance.

HEART SOUNDS IN GENERAL PARALYSIS

OF THE INSANE.

By J. MILNER FOTHERGILL, M.D., M.R.C.P.

A WORD, by way of preface to this paper, may not be out of
place. It takes its origin in no preconceived opinion, but
arose simply in this fashion. In October last, when at the
West Riding Asylum, Dr. Crichton Browne suggested to me
to examine the hearts of several patients who were the
subject of general paralysis, to see if, perchance, anything
could be found which might tend to throw light on that weird
disease. Without any expectation of what we should find,
twenty-two patients were examined, and a distinct accentu-
ation of the aortic second sound was found in seventeen of
them, and less distinct in one; making a total of eighteen
out of twenty-two. This accentuation was clear and dis-
tinct, and was as readily detected by Dr. Crichton Browne
and Dr. Bell Pettigrew as by myself. The proportion was
sufficiently large to warrant our supposing that it was
somehow connected with the disease, and not a mere coin-
cidence.

On inquiring into the literature of general paralysis I
found that Ernst Salomon regarded it as in many respects
resembling Bright's disease, i.e., that it is a constitutional
affection in which the starting-point is the brain and not
the kidneys. This was the more interesting in that the

Germans hold accentuation of the aortic second sound ('Verstärkung des zweiten Aortentones') to be one of the best marked physical signs of chronic renal disease (diffuse nephritis). That such a peculiar sign should exist in two chronic conditions, both especially associated with maturity in man, and in hale and strong men too, was a fact not only interesting in itself, but one which pointed distinctly to a condition in which the vascular system was decidedly implicated. In order to make further observations on the subject, Dr. Crichton Browne kindly gave me an opportunity of examining all the general paralytics in the West Riding Asylum. The condition of each is given in the accompanying notes, which state the salient points as briefly as possible.

As much care as could be taken was exercised in making these observations, in order to obviate the possible sources of fallacy arising from taking a series of lunatics in turn from the wards, some of whom were doubtful as to my intentions towards them.

The letters C. B. mean that the patient was under treatment by the Calabar bean, and this was noted, as a possibility existed that it might be found to exercise some control over the physical condition.

1. Thomas P—, 3rd stage, C. B. : 1st sound, somewhat sharp and short ; 2nd sound, slightly accentuated.

2. John L—, 3rd stage, C. B. : 1st sound, clear ; 2nd sound, not accentuated.

3. Henry S—, end of 3rd stage, C. B. : 1st sound, clear ; 2nd sound, accentuated and ringing.

4. John H—, end of 2nd stage : 1st sound, clear ; 2nd sound, accentuated, but not markedly so.

5. William N—, 3rd stage, C. B. (Othhæmatoma) : 1st sound, clear and thin ; 2nd sound, distinctly accentuated.

6. Henry T—, 3rd stage, C. B. : 1st sound, thin and clear ; 2nd sound, not markedly accentuated.

7. John F—, 2nd stage : 1st sound, thin and clear ; 2nd sound, accentuated.

8. Joseph C—, 3rd stage, no C. B. : 1st sound, thin and clear ; 2nd sound, not markedly accentuated.

9. Samuel W—, 3rd stage, C. B., Bronchitis : all sounds obscured.

10. Thomas W—, 3rd stage, Othhæmatoma : 1st sound, full and clear ; 2nd sound, accentuated.

11. Henry B—, 4th stage : both sounds clear and distinct ; very much alike.

12. Benjamin R—, 4th stage, Othhæmatoma: 1st sound, sharp and short, but clear; 2nd sound, slightly accentuated.

13. Eli D—, 2nd stage (excited): patient too noisy and violent to permit of any observations being made.

14. Daniel M—, 2nd stage, C. B.: 1st sound, normal; 2nd sound, accentuated.

15. Charles Edward T—, 3rd stage, has had C. B.: 1st sound, short and clear; 2nd sound, accentuated.

16. Joseph B—, 3rd stage: 1st sound, thin and clear; 2nd sound, distinctly accentuated.

17. Thomas D—, 3rd stage: 1st sound, full and clear; 2nd sound, accentuated.

18. James M—, 3rd stage: sounds quiet, and not marked.

19. George A—, 3rd stage: 1st sound, thin and sharp; 2nd sound, accentuated.

20. Hannah E—, about to be discharged, recovered: 1st sound, thin and clear; 2nd sound, distinctly accentuated.

21. Harriet E—, 2nd stage, C. B.: 1st sound, full; 2nd sound, somewhat accentuated.

22. Mary Ann B—, recovering, C. B.: 1st sound, thin and clear; 2nd sound, well accentuated.

23. Sarah Ann II—, 2nd stage, C. B.: 1st sound, full; 2nd sound, well accentuated.

24. Hannah G—, 3rd stage, C. B.; 1st sound, nearly inaudible; 2nd sound, loud and very distinct.

25. Jane E—, 3rd stage, : 1st sound, thin and clear; 2nd sound, accentuated.

26. Mary Ann C—, 3rd stage, C. B.: 1st sound, full; 2nd sound, normal.

27. Elizabeth B—, 2nd stage, C. B.: 1st sound, full; 2nd sound, normal.

28. Ellen W—, 3rd stage, C. B.: 1st sound, thin and clear; 2nd sound, distinctly accentuated.

29. Ann P—, last stage, C. B.: 1st sound, thin and clear; 2nd sound, accentuated.

30. Hannah H—, last stage, C. B.: 1st sound, thin and clear; 2nd sound, accentuated.

31. Mary Ann S—, 3rd stage, C. B.: 1st sound, thin and sharp; 2nd sound, distinctly accentuated.

32. Mary L—, 3rd stage, C. B.: 1st sound, sharp and thin; 2nd sound, accentuated.

33. Mary Ann W—, 4th stage, C. B.: 1st sound, clear; 2nd sound, markedly accentuated.

34. Alice H—, 2nd stage, recovering, C. B.: 1st sound, full and clear; 2nd sound, accentuated.

35. Martha S—, 3rd stage: 1st sound, clear; 2nd sound, well accentuated.

36. Margaret H—, 2nd stage: 1st sound, clear, but thin; 2nd sound clear and accentuated.

37. Martha O—, 2nd stage, C. B.: both sounds low and indistinct.

38. John S—, 2nd stage, C. B.: 1st sound, good; 2nd sound, very distinct.

39. Peter G—, 2nd stage, C. B.: 1st sound, thin and clear; 2nd sound, loudly accentuated.

40. David H—, 3rd stage: 1st sound, thin and clear; 2nd sound, distinctly accentuated.

41. Charles S—, 2nd stage: 1st sound, thin and sharp; 2nd sound, sharply accentuated.

42. William J—, 2nd stage, C. B.: sounds muffled; room noisy.

43. Joseph F—, 2nd stage: sounds normal.

44. William R—, 2nd stage, C. B.: 1st sound, clear; 2nd sound, accentuated.

45. Joseph P—, 2nd stage: 1st sound, normal; 2nd sound, well accentuated.

46. Thomas T—, 2nd stage (advanced): 1st sound, full and clear; 2nd sound, distinctly accentuated.

47. George J—, 2nd stage: 1st sound, thin and clear; 2nd sound, good accentuation.

48. Robert H—, 2nd stage, C. B.: 1st sound, clear; 2nd sound, accentuation good.

49. Harrison H—, 2nd stage, C. B.: 1st sound, thin and clear; 2nd sound accentuated.

50. William C—, 2nd stage: 1st sound, good; 2nd sound, accentuated.

51. James M'C—, 2nd stage, C. B.: 1st sound, thin and clear; 2nd sound, no accentuation.

52. William S—, 2nd stage: 1st sound, clear; 2nd sound, accentuated.

53. Peter S—, 2nd stage, C. B.: 1st sound, full; 2nd sound, abnormally distinct.

54. William B—, 2nd stage: 1st sound, thin; 2nd sound, abnormally distinct.

55. John R—, 3rd stage: 1st sound, clear; 2nd sound, distinctly accentuated.

Thus we find that of 55 observations, three (Nos. 9, 13, 42) were imperfect, and in 8 (Nos. 2, 11, 18, 26, 27, 37, 43, 51) the second sound was normal, or not sufficiently loud to be fairly termed accentuated. It must further be borne in mind that by accentuation is meant here such an increase in the sound as made it at once audible, and distinctly so, over the right costo-sternal articulation. This may fairly be termed accentuation, and all doubtful cases were rejected. Indeed, all care was exercised to make the observations correct, and to shut out all sources of error. No foregone

conclusions nor ready-made hypothesis existed, by which the facts might have been warped or moulded. The possession of these facts was, indeed, somewhat perplexing, as it was scarcely likely that a physical sign found in so large a proportion of cases (in 44 out of 55 cases) could exist without some distinct relation to the condition along with which it was found.

It seemed necessary to begin by exclusion. There was no reason to suppose that this accentuation was connected with aneurismal conditions of the aorta, though 'ringing' is the expression used by Dr. Warburton Begbie in his paper on Accentuation of the Second Sound, ' Edin. Med. Journal,' 1863 ; and indeed his expression 'ringing boom,' falls in with my experience of the sound as occasionally heard in the best marked cases.

It seemed that we must seek the explanation in one of these two conditions—1. Alteration in the valves themselves ; or, 2. An altered condition of the peripheral vascular area.

There seemed to be no reason to assume the existence of the first, and, consequently, the explanation must lie in the second possible cause.

This, again, might depend on two conditions—1. Increased arterial tension ; and 2. Increased vascular area.

Against the first was the fact that there is no distinct hypertrophy of the heart in general paralysis ; and also there was an absence of the firm and sustained pulse, the hard pulse, in fact, of increased arterial tension, such as characterises chronic Bright's disease. Though sphygmographic tracings, of which one will be given afterwards, would indicate some increased arterial tension, there is no evidence that it exists to so great an extent as to give rise to such distinct accentuation of the aortic second sound.

It became necessary, then, to examine the second possible explanation of it.

There was no evidence from the pathological anatomy of general paralysis that the vascular area generally is increased, but there were strong grounds for assuming that the vascular area of the cerebro-spinal system was enlarged ; and that, in

fact, the aortic valves were closed by a larger, and therefore heavier, column of blood *above* them, and that this led to the accentuation.

If this hypothesis was correct, it was obvious that a similar accentuation should be found in those cases of mania in which there is also cerebral hyperæmia ; and further, that some difference should be produced by posture, i.e., that this accentuation should be diminished when the patient was placed in the recumbent posture.

Accordingly, another series of observations were made to ascertain these two points.

We will take five cases of sub-acute mania first.

1. Joseph C—, pupils large, room darkened; pulse full, as if of dilated arteries; 1st sound normal ; 2nd full and loud, abnormally so (confined to bed).

2. John W—, pupils rather large ; pulse 72 (very quiet day with him) ; 1st sound normal ; 2nd loud and full (not so loud when lying down).

3. Joseph L—, pupils normal ; pulse 102, of medium strength ; 1st sound clear ; 2nd loud and full ; not much affected by posture, still the sound is not so distinct and loud when lying down.

4. George O—, pupils large ; pulse 78 ; 1st sound full ; 2nd full and loud ; distinctly affected by lying down.

5. Tom N—, pupils dilated ; pulse 102 ; 1st sound sharp and clear ; 2nd accentuated (2nd sound here like that of G. P.'s).

Here was found also the accentuation of the second sound, but it will be observed that a different expression is used in relation to it than to the sound in general paralysis, except in Thomas N—'s case.   In mania the accentuation was harder and more like the contact of two hard substances than the ringing sound of general paralysis.   In four of these five cases the sound was affected by posture ; in C—'s case it was not possible, or at least desirable, to put him up in an erect posture : so that in the four cases that could be tested, in all a difference, greater or smaller, was found produced by posture.   And it may be as well to state here that every care was taken to maintain the *bona fides* of the observations, and to see that the difference was not due to the inconvenience of listening to the heart of a man on a low bed, as compared to the unrestrained freedom of the erect posture.

Six other cases of general paralysis were then examined.

56. Thomas L—, 2nd stage, C. B.; sound natural; no accentuation of 2nd sound; no difference when lying down.

57. Christopher W—, 2nd stage, C. B.; pupils normal; pulse 90, good; 1st sound normal; 2nd sound loudly accentuated; altered when lying down, again distinct on sitting up.

58. James E—, pupils normal; pulse 105; 1st sound clear; 2nd sound ringing; sounds little affected by lying down.

59. William G—, pupils small; pulse 72, small; 1st sound thin and clear; 2nd sound distinctly accentuated; not so distinct when lying down as when standing up.

60. John D—, pupils small; pulse 54; 1st sound thin and clear; 2nd sound loud and distinct (2nd sound delayed); unmistakably affected by posture.

61. John R—, 3rd stage, no C. B.; 1st sound short and clear; 2nd slightly accentuated; not so distinct when lying down.

Nine cases previously seen were then re-examined in order to observe the effect of posture.

Henry S— (No. 2); pulse feeble; pupils normal; 2nd sound distinctly accentuated; much lost when lying down, again distinct when up.

John F— (No. 7), pupils small; pulse 94, good and sustained; 1st sound good; 2nd sound distinct and ringing; much altered by position, softer when lying down, again distinct when up.

William N— (No. 5), pupils somewhat small; pulse 112, quiet; radial artery tortuous and corded; 2nd sound loud and ringing; 1st sound sharp and thin; 2nd sound but little affected by position.

Robert H— (No. 48), pupils normal; pulse 84, very quiet; 1st sound good and full, disproportioned to pulse; 2nd sound loud and ringing; still distinct when lying down, but louder when up.

John R— (No. 55), pupils normal; pulse 90, quiet; 1st sound full and clear; 2nd sound accentuated and loud; more distinct when up than when in bed.

Joseph P—, pupils dilated; pulse 78; 1st sound good; 2nd sound not perceptibly altered by sitting up.

Hannah E— (No. 20), pupils somewhat unequal; pulse 108; 1st sound normal; 2nd sound accentuated; no difference perceptible on lying down.

Ellen W— (No. 28), pupils contracted; pulse 114, small; 1st sound sharp and thin; 2nd loudly accentuated; affected by posture.

Martha T— (No. 35), pupils unequal; pulse 78, small; 1st sound thin and clear; 2nd sound accentuated; not affected by posture.

Of these nine cases, five were affected by posture.[1] Thus in fifteen cases examined in order to observe the effect of posture in the accentuation of the second sound, in no less than ten was a difference found.

[1] In three of these cases some difference of expression is used as to the 1st sound, but none as to the 2nd sound.

So far, then, the hypothesis of the accentuation being due to an increased vascular area above the aortic valves was substantiated.

It may not now be out of place to see how far such a conclusion accords with what has been regarded as the pathology of general paralysis.

Though the etiology of general paralysis of the insane is far from a settled matter, there seems little doubt as to its taking its origin in 'abusive functional activity' (Dr. Crichton Browne.) A sufficient agreement exists among authorities to admit of my quoting the following passage from Niemeyer, when speaking of hyperæmia of the brain :—

'A fourth cause is paralysis of the vaso-motor nerves of the cerebral vessels. Physiological experiments show that if the cervical portion of the sympathetic be divided, the vessels on the corresponding side of the head become dilated. The cerebral vessels appear to be similarly affected by the use of spirituous liquors, by some poisons, as well as by great emotions and excessive mental activity. I would particularly call attention to the last cause, as I have frequently seen dangerous hyperæmia of the brain after too prolonged mental labour, which resulted fatally from the occurrence of œdema. We can hardly give any other explanation for these cases than that the walls of the vessels are paralysed by the above influences, their calibre dilated, and the supply of blood consequently increased.' [1]

That hyperæmia of the brain is found in general paralysis, in the earlier stage of mental exaltation, is universally admitted. The French writers have observed this clinically, and contrast it with the earlier stages of intoxication. Bayle wrote :—' L'ivresse, qui, au lieu d'être un état passager, deviendrait permanente, ne serait autre chose que l'aliénation paralytique. L'homme ivre est gai, content et heureux ; rien ne lui manque ; il a de la force, du courage, du talent ; souvent il se croit riche et opulent ; ses propos sont incohérents ; il est exalté et loquace ; en même temps sa langue

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[1] A curious corroboration of my view is found in this :—I examined a friend after long mental exertion, and found accentuation of the 2nd sound ; next morning, when fresh after a good night's rest, the accentuation was gone.

est embarrassée ; il prononce avec lenteur et en bégayant ;
sa démarche est mal assurée et vacillante. N'est-ce pas là le
tableau des paralytiques aliénés au premier degré ? Cette
comparaison nous paraît très-fondée.'

Bayle also taught that in many respects it resembled
chronic meningitis ; and L. Meyer, holding similar views,
found an increase of temperature during the paroxysms of
exaltation.

Different observers, as Rokitanski, Wedl, Salomon, and
Sankey, have described a condition of the vessels in the brain,
and especially in the cortical substance, as existing in general
paralysis, viz., dilated, elongated, kinked, twisted, and aneu-
rismal capillaries ; and, later on, a growth of nuclei in and
around the walls of the vessels, with subsequent contraction
and obliteration of the vessels, and increase of the connective
tissue or neuroglia of the brain, and enlargement of the peri-
vascular spaces of Robin and of His. This is really a condition
allied to that found in other viscera, viz., the liver, spleen,
kidneys, &c., where a condition of persistent hyperæmia is
followed by development of connective tissue and atrophy.
So in the brain we have a stage of hyperæmia with mental
exaltation, followed by a condition of atrophy, or brain
cirrhosis, where obliteration of the function of the brain
co-exists—a conclusion in harmony with the clinical history
of general paralysis.

This view is borne out by the ophthalmoscopic observations
of Dr. Clifford Allbutt, who ' detected a descending neuritis,
with first increased vascularity and engorgement, subse-
quently replaced by absorption of the exudation and extend-
ing whiteness of the disc, with, in most cases, atrophy.'
These observations of Dr. Allbutt's have been corroborated
by Dr. Aldridge at the West Riding Asylum.

The course of general paralysis, so far as it is connected
with vascular changes, is in accord with what we should à
priori expect. ' Where the hyperæmia is often repeated,
atrophy of the brain and decided dilatation of the vessels
result ' (Niemeyer). Before, however, proceeding to the later
stages of this affection, it may not be out of place to consider
the question of hyperæmia of the brain, associated as it is

with the explanation of the accentuation of the second sound adopted here.

Without adhering to the old idea of Kellie, Reid, Hughes Bennett, and others, that the skull is an inverted bell-jar, and so does not permit of any variation in the amount of its contents, or weighing the arguments and experiments of Burrows, &c., against it, it is certain that the brain is enclosed in a firm and unyielding case. This being so, there can exist no doubt but that increase in the bulk of oue of its contents is only attainable by a corresponding diminution of oue or more of its other contents ; for instance, arterial fulness is incompatible with venous fulness, and *vice versâ.* But there has been strong and irrefutable evidence adduced to show that the brain is more vascular under some circumstances than under others. This has been explained by supposing that there is a corresponding diminution and increase in the amouut of cerebro-spinal fluid. It is difficult to understand how this can go on in a bone-bounded space, except that the serous fluid is absorbed when the vessels dilate, and is effused again when they contract. Such an action is associated, in my mind, with the peri-vascular spaces described by Robin and by His. When the vessels dilate they fill the peri-vascular lymph-sheath or envelope, the lymph being absorbed ; and when they again contract, lymph fills the space betwixt the lymph-sheath and the bloodvessel. Westphal says, ' In the space between this envelope and the wall of the bloodvessel, there is discovered sometimes a clear fluid, intermixed with molecular nuclei, sometimes small, free, round corpuscles, likewise fat globules, and " hæmatosin," and quotes from Robin : " This envelope follows the course of the vessels, but, occasionally, at the point of their bifurcation, it does not form a corresponding angle, but becomes wider than usual, and presents a dilatation in which the bifurcation of the vessel lies. The nuclei in the intermediate space are sometimes few in number, sometimes numerous, but they do not touch one another." '

There is every reason to suppose that normally these lymph-spaces are but partially filled by the vessels, and that

when the brain becomes hyperæmic, either in consequence of stimulants, voluntary efforts, or vaso-motor paralysis, the vessels dilate and fill these spaces, the lymph being absorbed; the lymph again filling the spaces when the calibre of the vessels is diminished. Thus the small arteries and capillaries can contract and dilate within their lymph-sheaths, and thus the brain becomes more or less vascular, without interference with the other contents.

The combination of anæmia of the brain, and of œdema of it occurring together, is readily explained.

To quote again from Niemeyer's excellent article on ' Hyperæmia of the Brain,' ' When there is increased lateral pressure in the small arteries and veins of the brain, a transudation of serum from them into the peri-vascular spaces and interstices of the brain may very readily take place, and compression of the capillaries. It is only in yielding and distensible organs and tissues, which are not enclosed by firm envelopes, that any considerable œdema can co-exist with a normal fulness of the capillaries. In all tissues enclosed by fascia or other firm capsule, œdema causes anæmia of the capillaries.' Whether in the brain anæmia always precedes œdema is not very material; certain it is they are found together, and Traube states that œdema is the cause of anæmia, where there is spasm of the arterioles, and certainly in all fatal cases of anæmia in la Charité such œdema and anæmia are found. This view agrees with the occurrence of convulsions in the later stages of general paralysis when hyperæmia has given way to anæmia. These perivascular spaces, by depriving the vessels of the support they usually receive from the tissues around them, may exercise some effect over the frequency of rupture of the vessels in the brain (true apoplexy).

Where hyperæmia in a high degree has existed as a permanent condition, we should *à priori* expect that these perivascular spaces would be enlarged; and such a condition is actually found in the later stages of general paralysis. The long-continued distension has dilated the non-contractible lymph-sheath, and the space remains, while the atrophied and contracted vessel no longer fills it, and so there is a

large quantity of this effused fluid in the spaces of the brain
in chronic atrophy.   By the kindness of Dr. Herbert Major,
a microscopic section of chronic atrophy of the brain, de-
monstrating this well, is given here.

Section showing peri-vascular space with shrunken vessel within it.[1]

There is also a growth of connective tissue resulting from
this hyperæmia in the vessels or in the brain, and the cor-
puscles developed from the hyperæmia may even be ' colloid,'
or ' amyloid,' as shown in Salomon's paper.[2]   Lockhart
Clarke has found in general paralysis that these envelopes
around the peri-vascular spaces are not unfrequently darker
and more distinct than in the healthy organ.

The same observer made a curious observation in a section
of the brain of an old drunkard, where he found the peri-
vascular spaces distinct even to the naked eye, the section
looking like a section of Gruyère cheese in miniature.

A curious condition is also not unfrequently found in
general paralysis, viz., othhæmatoma.   Even if this be not
a hæmorrhagic inflammation of the cartilage of the ear,
due to vaso-motor paralysis, but due, as Gadden says and
Griesinger believes, to violence sustained in the wild-out-

[1] Drawn by J. C. Galton, M.A.
[2] ' Journal of Mental Science,' vol. iii. p. 365, 1862.

breaks of this disease, still some vaso-motor paralysis seems essential to its production; such hæmatoma not occurring in school-boys whose ears are also frequently exposed to violence.

In what relation, then, do these changes stand to the observations made on the altered heart-sounds. They bear out strongly the hypothesis of an increased vascular area above the aortic valves being the cause of the accentuation of the aortic second sound. The closure of the valves by a larger and consequently heavier column of blood than the normal one, would give a louder sound, if that sound be produced by closure of those valves, and it is now universally acknowledged to be so. The weight of the column is obviously in proportion to its size, and thus when the peri-vascular spaces are filled by the contained vessels, the weight of the blood-column is increased; its weight diminishing again when the vessels contract, by the amount of fluid effused—the lymph outside the vascular walls not adding to the weight of the blood-column. Thus the blood-column is heavier in cerebral hyperæmia in proportion as the vessels fill their peri-vascular spaces, and in proportion to the amount of lymph absorbed to permit of such dilatation.

When accentuation of the second sound is due to increased arterial tension, as in chronic Bright's disease, it is accompanied by hypertrophy of the left ventricle, often of a massive character, in order to overcome the resistance offered by this increased arterial tension; but in general paralysis no marked hypertrophy could be detected. In the second stage, the first sound, speaking generally, was fuller than in the third stage, as if the muscular wall were thicker than in the last stage; but this may not be more than that the heart alters with the general condition, there being general muscular plumpness in the second stage, and marasmus usually in the third and last stages. From the accompanying sphygmographic tracing it appears that some increased arterial tension exists in general paralysis, at least, if polycrotism be an indication of increased arterial tension.

The accentuation which is still found in the later stages of this affection is due probably to obstruction offered to the

blood-current by the atrophy of the blood-vessels in the head, and, according to Salomon, in other viscera. The pathological conditions in general paralysis are in unison with the
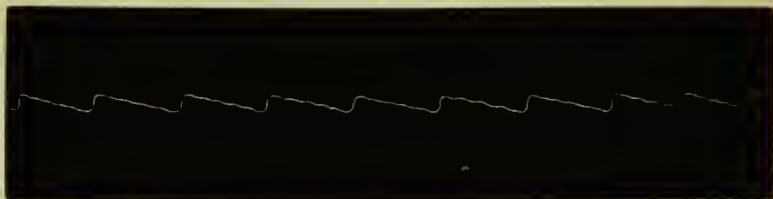
Sphygmographic tracing showing pulse form in general paralysis.

heart sounds, and the vascular changes of this condition are corroborated by the observations on the heart sounds. Still there is probably something more than the mere vascular changes in the different forms of chronic insanity, and on these more subtle changes the difference in the form of insanity depends; but the changes in the vascular system are of high importance, especially in the indications for treatment.

Such vascular changes are scarcely likely to proceed without some effect on the ganglionic nervous system; and such effect actually takes place. A general sclerosis of the sympathetic is found with a deposit of pigment, and this is chiefly found in the cervical ganglia. Adipose cells are also found substituted for nerve cells, and these two are also deeply stained with pigment. MM. Poincaré and Bonnet write: ' Les ganglions cervicaux sont considérablement hypertrophiés et offrent à la coupe les caractères du tissu squirrheux.'

It would seem as if a stage of hyperæmia in the ganglionic nervous system resulted in development of connective tissue; but whether some subtle change in the vaso-motor system precedes and affects the cerebral vaso-motor paralysis, or whether vaso-motor paralysis leads to these changes in the ganglionic nervous system, it is impossible to say. Whichever comes first in the order of time, both are found together at last.

Whether the different forms of insanity with grandiose delusions are the same from the first, as Bayle thought, or,

as Esquiral thought, simple insanity verges ultimately into general paralysis, cannot be determined, at least here. Baillarger proposes to class them together at first as ' congestive insanity ; ' however this may be settled, there is unquestionably a stage of hyperæmia of the brain, and an accompanying accentuation of the aortic second sound, at least in the majority of cases as yet examined.

This brings us to the broad question of the relation of heart disease to insanity.

On first starting this enquiry a strong impression existed that heart disease and insanity are largely associated. But, as a matter of fact, in all these observations only one murmur was heard, and that possessed all the characters of an anæmic murmur. Indeed, it would appear that as regards forms of insanity associated with cerebral hyperæmia, heart disease is so far an exception as almost to afford a protection against such an occurrence. It would appear that such a condition as sustained cerebral hyperæmia is incompatible with an ineffective or disabled heart. Griesinger, on ' Mental Diseases,' p. 194 (' Syd. Soc. Trans.'), says that the importance of heart disease in the production of insanity was much overrated by Nasse and others in the older psychological literature. And Griesinger might quite truthfully have added that, when Nasse wrote (1810), the diagnosis of heart disease was so imperfect that no reliance can be placed upon observations then made as to the condition of the heart. Griesinger gives some statistics, and sums up thus : ' According to these figures, we may assume that affections of the heart are rather rare than frequent in the insane.'

So far as my experience extends it would go to corroborate this conclusion of Griesinger's, and to point to an antagonism betwixt heart disease and insanity associated with cerebral hyperæmia. What the relation of heart disease may be to insanity depending on anæmic conditions of the brain is a subject which will require special investigation, and must form, in the future, a sequel to this paper.

As regards the present investigations, we may sum up in three brief but comprehensive conclusions—

1. That in general paralysis and in other conditions of cerebral hyperæmia, the heart's aortic second sound is usually accentuated.

2. That there is evidence pointing to the conclusion that this cerebral hyperæmia is connected with the peri-vascular lymph-spaces.

3. That a species of antagonism would appear to exist betwixt heart disease and insane conditions, associated with cerebral hyperæmia, and manifesting mental exaltation.

While heart disease is somewhat rare among the insane, disease of the cerebral vessels is in a different ratio. Vaso-motor paralysis, by exposing the elastic wall to the full force of the blood-distension (normally the muscular coat being in a state of partial contraction), leads to atheroma in the internal and external non-contractile layers, and consequently atheroma of the cerebral vessels is somewhat frequent. Hitchman found in 94 autopsies at Hanwell, and that, too, of insane women—not the sex with which atheroma is ordinarily associated—atheroma of the cerebral vessels in no less than 37 cases.

POWER OF PERCEIVING COLOURS

BY THE INSANE.

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Introductory.

WHEN it was suggested, a comparatively short time ago, that I should make this the subject of a paper for the present volume of Reports, the proposal was at once acceded to, for it had often appeared to me that some exceedingly interesting, if not highly practical, results might be obtained by honest and laborious investigation. From numerous conversations with those engaged in our speciality, it soon became evident that no one had ever examined the insane with the object of arriving at definite information as to their power of distinguishing colours. If the truth must be told, it must be confessed that very few persons indeed appeared to possess anything beyond the most hazy ideas as to colour-blindness and the many difficult and important questions raised in its consideration. Few had ever heard of acquired colour-blindness. They had never attempted to ascertain whether, when an insane person made a mistake regarding the colour of an object, the blunder was due to a real defect of vision or to mental disturbance. Indeed, it has evidently been the custom to take it for granted that insane patients always see colour normally, and no allowance has been made

for the possibility, in any given case, of the person being more or less insensible to colour. The question at once presents itself—Have we been right in so doing ? The probabilities evidently are that we have been careless in this respect, for it would be absurd to suppose that the colourblind never become insane. Although at the present stage of my enquiries it would be rash to attempt to give numerical results, still, my investigations tend to show that all forms of brain disease, as seen in asylums, are, in a large proportion of cases, accompanied by a more or less marked diminution of power to appreciate colour.

It is necessary to state here that the present paper is only a preliminary one, containing chiefly a brief abstract of the opinions and cases recorded by previous observers, with some of the results obtained by myself. By pursuing a new method of investigation, I shall be able to give next year an absolutely accurate account of all the cases examined, even when mistakes are made in the more delicate shades of colour. These results, I anticipate, will be of the highest interest. In the meantime, from following the methods of investigation hitherto chiefly employed, it is possible to record results interesting, though less complete.

To examine a lunatic on colour is a task beset with difficulties. When Dr. George Wilson was conducting his enquiries into chromatopseudopsis in Edinburgh, his patience was sorely tried by the students, soldiers, and others whom he examined. He found that intelligent men, unless they had followed some occupation conversant with colours, could seldom mention by name more than five or six. When requested to state the colour of various skeins of worsted, his pupils and others seldom employed any terms except red, blue, yellow, green, and rarely brown. Purple was scarcely ever named, and orange never. When we find such an imperfect knowledge of the nomenclature of colour among persons of at least average intelligence and education, we cannot be surprised to find that the patients in this asylum, drawn as a rule from colliery and manufacturing districts, know even less of the subject. As might have been expected, the examination of the women, when compared with that

of the men, was a very easy matter. A great deal of trouble was taken with the thorough investigation of the male patients, for among them it was expected that the most interesting results would be obtained.

Perhaps the greatest difficulty encountered by one investigating colour-blindness is the collection and examination of the literature of the subject. A very large proportion of the papers are contained in publications now seldom heard of, some of them are to be found in the libraries of the universities and scientific societies; but most of them cannot now be got anywhere. But these great libraries are almost inaccessible to a worker residing in the provinces, and it is only by means of much labour and searching that one can gradually accumulate a sufficient number of works on the subject to obtain a correct idea of the work done by former observers. Such a state of matters places the provincial worker at enormous disadvantages; for it may happen, and did actually occur in my own case, that whilst actively prosecuting his researches, he may procure a work which affords new information, shows that his method of investigation is imperfect, or that in the cases already examined some important point of enquiry has been omitted; in any of which circumstances he discovers that he has lost much valuable time and labour, and has to begin *de novo*. From these circumstances it is evident that this paper must be imperfect in many respects, in spite of all my exertions to collect original authorities on the subject. Such, however, as I have succeeded in procuring have been very carefully perused, and the leading facts and opinions in each extracted and subsequently arranged; so that in the following section of my paper I attempt to give a fair *résumé* of the whole subject of colour-blindness. The works of Wilson, Wartmann, Galezowski, Goubert, and a few others have been the principal sources of my information.

General and Introductory Remarks on Colour-Blindness.—Although colour-blindness is a subject which has been neglected to a wonderful extent by the mass of physiologists and men of science, it has been studied most attentively by

a few, so that at the present time its literature is very ex-
tensive, dating so far back as 1777, when Huddart published
a paper in the 'Philosophical Transactions of London.'   It
may be said, however, that Dalton was the first, in 1798, to
attract special attention to the subject by recording his own
peculiarities of vision.   He has indeed so identified himself
with it, that colour-blindness is perhaps more frequently
known as Daltonism.   English writers, especially Brewster
and Wilson, have been loud in their complaints that conti-
nental philosophers and writers should thus immortalise a
man by associating his name with a personal defect. In this
affair we have been perhaps rather sensitive, for Dalton him-
self does not appear to have been peculiarly ashamed of his
defect of vision, but rather the reverse ; for it is well known
that he often amused his friends in private by exhibiting to
them his peculiarities of sight ; and he at length published
a very full account of his own case.   We are moreover in the
constant habit of associating the name of a writer with any
subject by which he has made himself famous; and this is done
as a compliment to him.   We thus use the terms Bright's
disease, Addison's disease, and so on, without intending to
exhibit any want of respect to these celebrated men.

To obtain a fair idea of the literature of colour-blindness
for practical purposes, it is not necessary to collect and ex-
amine all contributions to the subject since the days of
Dalton ; for each writer has, as a rule, most carefully given
the results of previous observers.   We have thus brought
down, in the more recent papers, most of what is important
in the writing of the earlier investigators.   It must also be
remembered that in Dalton's time, and for many years after-
wards, the physiology of vision was but very imperfectly
known ; and by far the most important additions have been
made to our knowledge of the functions of the retina since
the introduction of the ophthalmoscope.   When Wilson wrote
in 1854, he stated that he had then for the first time been
enabled to view the retina in the living eye by aid of the
ophthalmoscope, and he evidently had the idea that this in-
strument would be useful in furthering the researches of
those interested in colour-blindness.   What he expected has

been fulfilled, but in a very different manner than he anticipated.

In our desire to utilise the results obtained by former observers, it is most important to remember that many of the older recorded cases are almost entirely useless for our purpose. The methods of examination employed were so imperfect, and the results were described in such loose and uncertain terms, that they are really of no scientific value. More modern writers have caused themselves infinite trouble in attempting to incorporate these observations with their own; they have tried hard to classify them and to explain the phenomena stated to have been observed. The only result of this misdirected energy has been to complicate the whole matter. This is greatly to be regretted when we remember that these observations which have caused such trouble to subsequent observers were very imperfectly investigated and recorded.

Definitions and Denominations of Colour-Blindness.—According to the views entertained, very different names have been applied to the affection under consideration; indeed, opinions have differed much as to what should be considered true cases of Daltonism. Chrupsia, or coloured-vision, may exist as a permanent disorder, or may occur during the course of other diseases or as the result of substances administered to the patient. This disease, though one in which there is a disordered perception of colour, must not be confounded with true colour-blindness. Mental emotion causes a transient form of chrupsia; for persons when greatly alarmed see surrounding objects of a grey or blue colour. The most familiar example of coloured vision occurs in jaundice, and it is well known that by the administration of santonine our perception of colour may be so much altered that all surrounding objects appear yellow. True cases of colour-blindness differ entirely, however, from these. In a genuine example, the person is unable, to a greater or less extent, to perceive the colour of objects, whilst his perception of form remains perfect. Wilson relates the case of an engraver absolutely colour-blind, who was most expert at his profes-

sion, and, indeed, found his defect of sight of advantage to him in certain circumstances.

Authors appear to have exercised great ingenuity in inventing barbarous names for colour-blindness. In their attempts to give a complete definition of the affection in a single word, they have but very partially succeeded, but their success has been remarkable in the number of evil-sounding words which they have introduced into our scientific vocabulary. Of the numerous denominations of colour-blindness it is only necessary to reproduce a few, as most have been invented by writers whilst entertaining mistaken views of the real nature of the affection. *Achromatopsis* is really a misnomer when applied to all cases, for the word really implies the impossibility of perceiving colour. *Chromatopseudopsis* is employed by Wilson and others; Goethe, from the belief that the non-perception of blue explains the phenomena of colour-blindness, uses *Akyanoblepsis*. Wardrop speaks of *coloured vision*, which term is more applicable to Chrupsia, or coloured vision of colourless objects: Ruete employs the term *Anerythroblepsis*; and we have in more or less use, and with meanings more or less definite, *Chromatometablepsis, Daltonism, Heteropsis, Dyschromatopsis, Parachromatism, Pseudopsis*. Sommer and Szokalski suggest Chromatopseudopsis. Sous adopts Dyschromatopsis, as proposed by Taylor. To these already given we may add, as remarkable specimens of scientific nomenclature, *Chromatodysopsis, Dyschrosis, Pseudochromia*.

It is to be regretted that some recent writers, as Goubert, have insisted that true colour-blindness is necessarily congenital. Formerly, no doubt, this opinion was universal, simply because no one appears to have taken the trouble to look for cases which were not congenital. To insist on this point introduces additional difficulties. It must be remembered that it is not always possible to discover at what time of life the defect was first discovered in any given case. Many cases, regarded as genuine, are on record where the defect of vision was not detected until the person had attained adult life. It is also impossible to deny the possibility of colour-blindness sometimes being due to cerebral

causes occurring in early childhood. A well-marked condition of colour-blindness appeared in an adult as the result of a severe injury of the head; and it is within the range of possibilities that a similar condition may occasionally follow the numerous cerebral and other nervous diseases of childhood. It is only necessary to study the histories of recorded cases to see at once the great difficulty which besets this portion of the subject; and as no real advantage is to be derived from this manner of viewing the matter, it is, perhaps, better not to insist that any given case must be proved to be congenital before it is recognised as genuine.

As defined by Goubert, colour-blindness is a congenital, permanent, and hitherto incurable affection of vision, without disorder of the refracting media of the eye, without lesion recognisable by the ophthalmoscope or by *post-mortem* examination with the microscope; consisting in a total or partial inaptitude to receive the impression of colours, and, consequently, of distinguishing the one from the other. He, as we have already mentioned, insists upon the affection being congenital, and upon the absence of material lesions; for he does not recognise as achromatopsis, properly so-called, the accidental, acquired, temporary, or symptomatic cases with ophthalmoscopic disorders. They belong to pathology, whilst, in his opinion, true colour-blindness always belongs to the domain of physiology.

Classification of the Colour-blind.—When Wartmann wrote his paper in 1840, he stated that the only observers who had at that time attempted to classify Daltonians were Seebeck, Szokalski, and Purkinje. Seebeck arranged them in two divisions. The first comprises those individuals who are more deceived in the degree of colouring than in the nature of the colours. The tints which they confound more or less are,—light orange and pure yellow; dark orange, light yellowish or brownish green, and yellowish brown; pure light green, grey, brown and flesh colour; rose red, green of a more blue than yellow tint, and grey; crimson, dark green and chestnut brown; bluish green and dirty violet; lilac and blue grey; azure, blue grey and lilac grey. Their sense is very defective for the specific impression of all

the colours in general ; it is especially so for that of red, and consequently for that of green, which is its complementary tint—colours which they distinguish little, if at all, from grey ; it is further defective for blue, which they distinguish very incompletely from grey. Their appreciation of yellow is the most correct, although they often see less difference between it and the appearance of colourless bodies than is the case with the ordinary eye.

The second division of Seebeck comprises those persons who confound light orange, greenish yellow, brownish yellow, and pure yellow ; bright orange, yellow brown, and grass green ; brick red, rust and dark olive green ; flesh colour, grey brown, and bluish green ; dull bluish grey, and grey a little brownish ; dirty rose, somewhat yellowish, and pure grey ; red rose, lilac, azure and grey passing into lilac ; crimson and violet, dark violet and dark blue. They have only a feeble power of perception of the least refrangible rays ; this is their most strikingly distinctive character. Lastly, yellow is the colour which they recognise best ; they distinguish red objects a little better, and blue ones a little less than colourless bodies, but above all reds from blues in a much less decided manner than persons of the first class.

Szokalski in 1842, adopting the denomination of chromato-pseudopsis for defective cases of colour-perception, divided them into the five following classes :—

1. That of persons in whom the sense of colour is almost completely wanting, and who, in place of the elementary colours, yellow, red, and blue, see only different degrees of white and black.

2. That of persons who not only see yellow, but are besides capable of a particular perception, and the same for blue and for red. These are the Akyanopes of Goethe.

3. That of persons who also distinguish yellows. External objects appear to them coloured with shades which generate the different mixtures of yellow, of white, and black.

4. That of persons destitute of the perception of red, which appears to them ash grey.

5. That of individuals who distinguish all colours, but not in a decided manner; instead of being able to distinguish the mixture of two colours, they never see but one of them.

Purkinje divides the colour-blind into four classes, which he denominates Achromatopsis, Chromatodysopsis, Akyano-blepsis, and Anerythroblepsis. Of these terms the first two relate more to the intensity, the others to the nature, of the imperfection.

Wartmann is of opinion that the weight of evidence is in favour of Seebeck's classification. Nevertheless, whilst urging the necessity for a strict classification of the colour-blind, he professes to throw aside all proposed classifications as failing to include all varieties of the disorder. These he believes to be as numerous as the individuals who suffer from it. He is therefore content to arrange all cases into two classes, according as they are dichromatic or polychromatic.

Without going into detail at present, it may not be out of place to give now the whole of Wartmann's conclusions as to colour-blindness. His paper on the subject is a very important one, is constantly referred to by most modern writers, and may, to a certain extent, be regarded as embodying all that was known on the subject up to his time. He concludes his paper by stating that he has attempted to establish the following facts :—

1. That Daltonism was not investigated by the ancients.

2. That among the moderns it has only been authentically proved in individuals of the white race.

3. That there exists a very considerable number of varieties of it, from those persons who only perceive two colours, or rather two sensations—the one of brightness, the other of obscurity—to those who even by candle-light confound any colours which closely approximate, such as shades of blue and green of equal intensity.

4. That there are many more Daltonians than is generally supposed.

5. That the female sex furnishes a very small proportion.

6. That they can, in certain cases, be recognised by some external signs.

7. That there are as many with blue as with black eyes.

8. That Daltonism is not always hereditary.

9. That it does not always affect all the male members of the same family.

10. That it does not always date from birth.

11. That Daltonians do not judge complementary colours as we do.

12. That several of them are not sensible to the least refrangible rays.

13. That they see, as we do, the lines of the spectrum discovered by Fraunhofer, consequently the limits of brightness and of interior obscurity of the spectrum.

14. That they do not judge as we do of the contrast of colours.

15. That their affection does not proceed from a vicious conformation of the eye, nor from any coloration of the humours of the retina.

16. That the state of Daltonism may be altered by means easily employed.

17. That it has its origin in a defect of the sensorium.

As an example of modern classification we may refer to that of Goubert. He makes the following groups :—1st. Persons who see only black or white. 2nd. Those who see only yellow. 3rd. Those who perceive only yellow and blue. 4th. Those who recognise yellow, blue, and red, but cannot readily distinguish their mixtures. 5th. The large number of persons who cannot appreciate delicate shades of colour, either at night or with the aid of sunlight.

Although Goubert puts this forward as a classification, he evidently has but little faith in its accuracy, for he acknowledges the great difficulty there is in attempting to lay down a hard and fast line between normal and perverted vision. He fears that in doing so he might run into the grave error, certainly committed by some writers, of exaggerating the numbers of the colour-blind. He also has observed, and Wilson directs special attention to the same fact, that in certain well-marked examples of Daltonism, individuals can perceive colours better at one time than another, and, which is a remarkable fact, that they can frequently see best with the aid of artificial light.

In their anxiety to manufacture a classification based upon power of perceiving colour, writers have quite overlooked the fact, so skilfully and beautifully pointed out by Wilson, that it is beyond our power to subject an eye simply to the influence of colour. His remarks on this subject are so impor- tant, and the difficulties suggested are so peculiar, and have yet attracted no attention, that we venture to make the fol- lowing somewhat lengthy quotation :—' And even if it [the retina] were altogether unimpressible by colour, it appears exceedingly doubtful whether we should be able to discover if it was. We have it not in our power to subject an eye simply to the influence of colour. Every luminous ray is (in the lan- guage of the material hypothesis of light) a bundle of colour- giving, heat-giving, and chemical or actinic rays, mingled in unequal proportion. Each colour-ray, therefore, carries along with it to the retina a different number of heat rays and chemical rays. Thus, if for simplicity's sake, we assume only three colours to exist, red, blue, and yellow, then the red ray of the spectrum, which the painter metaphorically styles a warm colour, is in reality much hotter, when tried by the ther- mometer, than the blue or yellow. The blue, which is the coldest and darkest of the three primary colours, greatly ex- cels the red and yellow in the number of chemical rays which accompany it ; whilst the yellow, which is totally destitute of such rays, and contains very few heat rays, must necessarily consist almost entirely of colour rays ; and it further excels red and blue in luminosity. The retina, accordingly, in so far as it is influenced by heat, will be most affected by the red ray ; in so far as it is susceptible of chemical change, by the blue ray ; and in so far as it is influenced by luminosity, by the yellow ray, apart from the special impressions made upon it by each ray in virtue of its colour.

' When, therefore, we ask any one to gaze at a rainbow (which I select as the great natural representation of many colours), and tell us what he sees in it, we ask him to report on a series of complex sensations, to the production of which the brightness, heat, colour, and chemical force present in the light by which he sees it, all do, or at least may, contribute. Such a spectator, though absolutely blind to colours, might

retain susceptibility to all the other influences of light, and would probably possess it in a higher degree than those who were not colour-blind. It is certain at least, as the sequel will show, that the colour-blind of all degrees often possess in perfection the power of distinguishing shades of the same colour; and that when they confound two colours, such as red and green, they assort together, with great nicety, the light and dark shades of the one with similar shades of the other.

' The most severe sufferer, accordingly, from colour-blind-ness may be expected to see as large and as perfect a rainbow or spectrum as others do, although to him it is colourless. The different bands, such as the bright yellow, the dark blue, and the intermediate red, will affect his eye differently in virtue of their different luminosity, and if he had been accus-tomed from early life to distinguish degrees of brightness by the terms yellow, red, and blue, we should never discover, by his description of the rainbow, that he was colour-blind at all. . . . But all cases of colour-blindness agree in this, that to the extent of its occurrence in any one, it implies a condi-tion of vision in reference to which there is not a common experience, and therefore cannot be a common language be-tween those conscious of colour and those unconscious of it. The information, accordingly, which they can convey to each other is almost solely of a negative kind. We cannot, for ex-ample, give to one who never saw green a positive conception of what we understand by it; we can at best make him aware that it is none of the colours he does see. And he on his part cannot make us understand what positive impression green makes upon his eye, although he may satisfy us that it is something different from that which blue or yellow makes.

' It must therefore be remembered, that the report of every case of colour-blindness is rendered hopelessly imperfect in a twofold way, viz., by the impossibility of subjecting the eye to the test of colour, unaccompanied by other agencies, and by the impossibility of procuring from the colour-blind a positive account of the peculiarities of their vision.'

In our preliminary remarks it is of course impossible to mention, much more to discuss, the many subjects connected

more or less closely with colour-blindness. We must therefore be content to glance at those of greater importance, and immediately bearing on our subject.

Frequency of Colour-blindness.—All observers agree that total colour-blindness, where all colours appear as shades of white and black, is very rare; indeed, some have been tempted to question the existence of such cases, but that they do sometimes occur may now be considered beyond doubt. On the other hand, however, the cases are extremely common in which the finer shades of the more composite colours are confounded together. The fine appreciation of colour is acknowledged to be a power possessed by comparatively few; as a rule, only by those who have been specially educated to do so. But in this the eye does not differ from the other organs of special sense. Between the ear and the eye there has been shown to exist a remarkable parallelism of function. It is a curious fact that some sounds quite audible to normal ears are not heard by others. Persons possessing this peculiarity fail to hear sounds when they pass beyond a certain pitch; when the note is either too shrill or too deep it becomes inaudible to them. The similarity of this condition to that of colour-blindness is evident enough, and it is quite possible that the thorough examination of each may be able to lead to the explanation of both. By early and skilful education the ear can be educated to detect wonderfully minute differences in the musical pitch of sounds; so the eye can be trained, as in artists, dyers, &c., to an acuteness in recognising variety of shades which is quite beyond the power of ordinary individuals.

As we have already had occasion to remark, it is extremely probable that the number of the colour-blind in the general population has been much over-stated through the faulty methods of examination. The popular nomenclature of colours is very imperfect, and, as already stated, extremely limited; consequently the more unusual colours are named according to their similarity to others better known. In examining persons, therefore, for the detection of colour-blindness it is absolutely necessary to avoid asking them to name colours submitted to them; at least some other means

must be adopted to guard against certain error. None of my patients willingly confessed their ignorance of colour; but, as a rule, the more ignorant and uneducated they were, the greater was the readiness with which they attempted to name the colours of the various skeins of worsted submitted to their inspection. A very considerable number of those whom I examined had recovered from their mental derangement, and were awaiting their discharge. Amongst these, therefore, it cannot be urged that their mental state complicated their examination, yet many of their answers displayed an ignorance of colour absolutely surprising. They had certainly managed to learn that grass is green, bricks red, and the sky blue; yet many of them confounded blue and green when asked to name the colour of pieces of cloth and paper; they called a blue paper green, and a green one blue, without appearing to be in the least aware of their error. Had their examination finished here, most erroneous conclusions would have been drawn from it; for the majority of these patients arranged wools according to colour and shade with great accuracy.

Confining attention to the more important class of the colour-blind, that in which red, yellow, blue, purple, orange, green, brown, &c., are respectively mistaken for other colours, or all confounded together, we find very various estimates as to the frequency of their occurrence. Dalton found on one occasion 12 per cent. of his subjects colourblind, on another 8 per cent. Wilson in his work has given full details of the estimates made by various writers. Prevost calculates from his observations that 5 per cent. of the population are Daltonians; whilst Seebeck found 5 out of 40 youths, who composed the two upper classes in a gymnasium at Berlin, the subjects of the same defect. Kelland found among 150 students 3 who could not distinguish red from green. As the result of his own observations Wilson found that 31 soldiers of the 4th Regiment of Infantry were colour-blind. Five confounded full red with full green, and 1 pink with light green; 13 confounded brown with green, and 12 blue with green. 14 men among 177 of the 7th Hussars were Daltonians; 4 mistook full red for full green,

and 1 pink for light green ; 2 brown for green, and 1 yellow for pink. Five of 123 artillery-men were marked cases of colour-blindness, 2 were doubtful. Two mistook full red and green, 1 brown and green, 2 blue and green, and 2 appeared to mistake purple and yellow. As the result of all his investigations he concludes that 5·6 per cent., or 1 in 17·7, are colour-blind; 1·8 per cent., or 1 in 55, confounded red with green; 1·6 per cent., or 1 in 60, confounded brown and green ; and 2·2 per cent., or 1 in 46, confounded blue with green.

External Appearance of the Eyes of the Colour-blind.—This is a matter of but little importance, but may be noticed in passing. It has been asserted by some, and as strenuously denied by others, that the appearance of the eye is, in many cases, characteristic. Wartmann has observed that in the colour-blind with hazel eyes there is a golden lustre of a peculiar tint. He attaches considerable importance to this supposed fact, because he imagines we might at once be able to detect the suitability of certain individuals for employments in which a knowledge of colour is necessary. Unfortunately I have not kept any notes of the colour of the iris in the patients I have examined, and am, therefore, unable at present to give any answer on another disputed question, viz., whether blue or black eyes are more frequent among those more or less insensible to colour.

Sex in its Relation to Colour-blindness.—All observers agree as to the much greater frequency of colour-blindness among males. Statistics on this subject, indeed, refer to them alone. That some well-marked cases have been observed among women is a well-known fact, but as yet we do not possess any reliable information as to their frequency. To remedy this defect in our knowledge of this part of the subject, Wilson has suggested that teachers in schools of design, surgeons in factories, and others, should settle the matter by examining those coming under their care. He, if I remember rightly, very shrewdly suggests that women may have some reluctance to admit such a defect of vision, and use every artifice to conceal it. Thus, a woman more or less

colour-blind would make her purchases with a female friend endowed with a perfect perception of colour.

Some few observations, it may be remarked in passing, have been made on colour-blindness in relation to race. These have been much too limited in number to admit of definite results ; but so far as they have gone they seem to indicate that the black races have a much more acute sense of colour than we possess.

Hereditary Character of Colour-blindness.—No fact is better established than that, in the very large majority of cases, this affection is hereditary. It is unnecessary, however, to enter at length into this question, as the laws which appear to govern the occurrence of colour-blindness appear to be very similar to those which exist in other diseases.

Theories concerning Colour-blindness.—Some of these I must pass over, as some most difficult questions in optics and mathematics would otherwise require to be discussed, a task for which I do not possess sufficient knowledge. Most of the theories suggested in explanation of the phenomena of colour-blindness may be arranged in two classes, the Chromatic and the Cerebro-retinal.

Dalton concluded that the media of his eyes and of those affected like himself were abnormally coloured, probably by some modification of blue. He supposed that it must be the vitreous humour, as examination of the eyes had failed to detect any tinting of the aqueous humour or lens. His theory, however, survived only until his own death. A most careful *post-mortem* examination was made, with the result of showing that his eyes in all their tissues were free from abnormal colouring.

Sir David Brewster observed, during the dissection of many hundred eyes of the lower animals, that the vitreous humour was in several instances of a greenish-blue colour. Writing in 1844, he offered the suggestion that a blue retina might be one of the causes of colour-blindness, ' but only on the supposition that the choroid should be proved to be the seat of vision.'

Wilson has considered at great length the relation of the general colour of the retina to colour-vision. He says that

it appears to him certain that, 'admitting the retina to be physically the seat of vision, its colour, if at all considerable, would render the perception of colour abnormal.' He attaches great importance to the colour of the yellow spot. By its colour he explains the fact that the human eye is more sensitive to yellow than to any other colour, and he suggests that 'the spot of Sœmmerring is a provision for securing to the human retina the conversion of white into the more exciting yellow light, which makes the maximum impression upon it.' He is accordingly forced to the conclusion that objects have different coloured appearances according as the eye has or has not, as is the case with many animals, a yellow spot.

Another most important question which he attempts to settle is, whether the light reflected from the eye has any influence upon the perception of colour. From the normal human eye the amount of light reflected does not appear to be great; but from the eye of an albino it is very considerable; and in the case of many of the lower animals whose eyes are furnished with tapeta, the reflected light is strongly coloured, being blue, green, and red according to circumstances. There do not appear to be any grounds for the supposition that in the human eye the perception of colour is affected by the rays of light reflected from the fundus. Wilson imagined that these rays, not having been absorbed by the choroid, but having to pass again through the retina, would act for a second time upon it, causing a sensation of light, but on this occasion of a more or less yellow colour, this result being due to the light having to pass through the retina containing the blood corpuscles in its capillaries. So far as observations have been made on albinos, there does not appear to be any foundation for the supposition that the light reflected from their eyes affects their power of perceiving colour. Besides, we have no reason to believe that light acting on the retina from behind can affect it at all.

We need not enter into the question, a very interesting one though it be, concerning the perception of colour by animals which have tapeta. The light reflected from their

eyes is brilliantly coloured ; in certain circumstances it assists to illuminate external objects, and it is obvious that the coloured perception of the same object obtained by any animal must vary according to the colour of light reflected from its own eye, be it green, blue, or red. Some believed that the green light reflected from the ox's eye was for the purpose of giving the grass on which it fed a more brilliant colour! Unfortunately, we cannot examine animals as to their perception of colour, but we can observe the effects of certain colours upon them. Taking the bull, ram, and turkey, as examples of animals easily excited by the spectacle of red, and as easily calmed by green, Wilson shows that each of these animals cannot receive a similar coloured sensation, for the ram's tapetum is much greener than the bull's, and the turkey has no tapetum at all.

Cerebro-retinal Theories of Colour-blindness.—Various have been the notions entertained as to the relative powers of the retina, and the various nervous centres with which it is connected by the optic nerve, in enabling us to perceive colour. Is there a special arrangement in the retina, or is it in the ganglia of the optic nerve that the influences from the optic nerve are so elaborated or modified that we are able to receive ideas of colour ?

Helmholtz has given in his adhesion to the theory first promulgated by Young, that there are three kinds of nerve fibres, each of which can be influenced only by one of the primary colours. There are special fibres for the red, fibres for the green, and others for the violet. Although Helm-holtz says that this theory gives a clear and simple explanation of all the phenomena of colour-perception, we have not yet proved by anatomical investigation that such special fibres as he speaks of really exist. Besides, even admitting the existence of these fibres, it cannot be said that all the phenomena of coloured-vision and colour-blindness can be explained by Young's hypothesis.

Various writers have attempted to account for the non-perception of colour by supposing some defect in the sensitiveness of the retina to the undulations of light. Wartmann, Sous, and others consider that the elasticity of the retina

has been diminished, the shocks resulting from the vibrations of various colours produce the same impression, and consequently they are confounded by the observer. Kelland also agrees with Wartmann in most points : they worked independently of each other, and arrived at conclusions almost identical.

In this very rapid sketch it would be wrong to omit the only theory of colour-perception which suggests the existence of a special organ of colour—we refer to the phrenological hypothesis. Gall and his followers place the organ of colour in that part of the brain situate immediately above the eye and beneath the eye-brow. Although most men may agree that there must be such an organ as the phrenologists maintain there is, perhaps few will consider the evidence sufficient to justify the position selected for it by that school of cerebral physiologists. So far as is known, Dalton is the only colour-blind person who had a *post-mortem* examination performed for the express purpose of discovering the material cause of his defect of vision. The examination was made in presence of Mr. Bally, a disciple and former assistant of Dr. Spurzheim, and he pointed out to those present a remarkable prominence on the frontal portion of the orbital plates, and the imperfect or deficient development of the convolution of the anterior lobes, which rested on them. It is more than questionable if this can be claimed as a case supporting phrenological views, for it is undoubted that, for seven years before his death, Dalton was an aphasiac. When 71 years of age he became paralysed on the right side, and his power of articulation was much impaired, as well as his memory of words. A detailed account of the condition of the brain is given in his biography, and it is quite evident that no conclusions regarding the seat of the organ of colour can be drawn from a brain otherwise so much diseased.

Colour-blindness among the Insane.—It was my intention to have noticed in this paper acquired colour-blindness, the circumstances under which it occurs, and the ophthalmoscopic appearances of the eyes in cases of congenital and acquired daltonism, and in the insane. But at present space does not permit of my doing so.

I have been unable to discover any recorded cases of insanity in which the power of perceiving colour was specially examined. Even in patients suffering from well-marked hallucinations of sight we do not find that any efforts have been made in this direction. But we have several cases of severe cerebral injury, followed by more or less mental disturbance, in which permanent colour-blindness remained after all other indications of cerebral mischief had disappeared. A case, reported by Wilson, is exceedingly interesting in this respect. We therefore give a condensed account of it.

A gentleman engaged in practice in Yorkshire was thrown from his horse in November 1849. He suffered severe injury of the head, was confined to bed for some months, and, upon recovery, had a very imperfect recollection of what occurred during that time. It appears, however, that after rallying from the collapse which immediately succeeded the accident, he suffered from severe pains in the head, delirium, mental excitation approaching almost to mania, loss of memory, and other symptoms of cerebral disturbance, which did not subside for many months. On recovering sufficiently to notice objects around him, he found that his perception of colours, which was formerly normal and acute, had become both weakened and perverted; and it had continued so up to the date of his examination by Dr. Wilson. To him the rainbow appeared quite destitute of hue, and as a white semicircle against the sky. Certain tints, however, of coloured objects held near to the eye were well enough distinguished, especially yellow and blue. He found bright shades alone pleasant to look at; dark shades appeared a mass of confusion, and uncomfortable to the eye. Red and green in all their shades were undistinguishable from each other. After a full and careful examination, it was found that he never mistook blue and yellow, always mistook red and green, and put aside, as incapable of definition, all the more mixed or composite colours.

I may now give shortly an account of my method of investigation and the results obtained.

About 120 skeins of worsted having been spread upon a

white table-cloth, the patient was requested to pick out a skein or two to match one in my hand. This process was repeated until I was satisfied that the patient had a correct appreciation of colour, or the contrary. All these examinations were recorded at the time, but patients whose condition called for investigation have been re-examined recently.

The great difficulty encountered was, when to put aside a patient as unreliable on account of his mental condition. Being determined not to err by exaggerating the number of the colour-blind, I fear that I have erred by rejecting some cases which were genuine cases of daltonism. However, this error does not so much signify at present, as I shall not attempt to give any definite statistical information on the matter.

Following Wilson's example, I considered a patient free from colour-blindness if he or she could tell the primary, secondary, and chief tertiary colours. In future examinations I shall, however, record the smallest errors committed with these hues. Among those allowed to pass as having answered correctly was a large number of men and women who confounded very dark green and dark purple with black. But after eliminating all doubtful cases, sufficient remain to prove that there is a much larger proportion of the insane more or less colour-blind than there is in the general population.

Attempts were made to examine 438 women. Of these 78 suffered from epilepsy; 22 from general paralysis; 118 from dementia; 8 from cerebral atrophy; 71 from chronic mania; 32 from mania; 24 from recurrent mania; 36 from melancholia; 9 from monomania; 6 from acute mania; 4 from subacute mania; 1 from hysterical mania; 1 from brain disease of uncertain form; 7 were imbeciles; and 21 were convalescent. Of these it was considered advisable to reject the answers of 114: they were, cases of epilepsy, 25; general paralysis, 5; dementia, 47; chronic mania, 12; mania, 5; acute mania, 4; cerebral atrophy, 5; recurrent mania, 4; melancholia, 4; and 3 were imbeciles. Now it is very important to remember that these patients were so ignorant of

colour that, had they been sane people, they would have been considered examples of colour-blindness ; but their state of dementia or excitement was such that it was determined to exclude them. But my observation of these cases leads me to the belief that the vast majority of these very demented people live without being conscious of the colour of external objects. At present I am unable to prove this conclusively, but I hope yet to bring forward observations which will show that such is the case.

The remaining 324 women were considered sufficiently intelligent that their answers might be relied on. Of these 319 certainly perceived colours normally ; but 9 were evidently more or less colour-blind. They were as follows : —

CASE 1.—E. K—, æt. 29.—Chronic dementia subsequent to a third attack of insanity. She has been in the asylum since November 1868, since which date almost no change has occurred in her condition. Her present state is one of well-marked dementia, but she still retains sufficient intelligence to answer questions and to understand exactly what is required of her.

She constantly confounds red and green ; matches pale pink with white. She never mistakes yellow. Dark green and very dark purple she always matches with black. Greens in which yellow predominates she invariably calls yellow, and with the more complex tints her mistakes are constant, and, so far as can be made out, her classification of these is made entirely at random.

CASE 2.—S. T—, æt. 38.—She is an epileptic, and has suffered from fits for the last ten years at least. Her appreciation of colour is greatly diminished. She knows light green and light blue well ; dark blues and purples she always arranges together, and classifies very dark green and purple with black ; all flesh colours with pink ; and light browns with yellow. She hesitates a long time before naming any colour, except the primary ones, and fails almost entirely in matching the complex tints.

CASE 3.—P. K—, æt. 27, labours under puerperal mania, of about four years' duration. At present she is too excited to conduct her re-examination satisfactorily. When first examined, she was much quieter, and understood what she was about. From hurried notes made at that time, it would appear that she confounded blues and greens. When this fact was discovered, she was set aside for further examination, which, however, it has been impossible to make, and I am therefore unable to give a full account of her peculiarities of vision.

CASE 4.—B. D—, æt. 18, labours under acute dementia, of about six months' duration. All dark greens and dark purples she arranges with black ; blues and light purples ; red-orange with a ruddy brown. She classifies with red russet, orange, and ruddy-brown. She knows the primary colours when she sees them, but confounds all sorts of complex colours with them.

CASE 5.—M. W—, æt. 45, labours under dementia, of twenty years' duration. Like many cases, she cannot tell any difference between very dark green, very dark purple, and black; all moderately dark purples she arranges with blue. Under red she arranges scarlet, crimson, pink, and red-orange. She appears quite unable to arrange the varieties of red as she does with blue and yellow.

CASE 6.—M. H—, æt. 27, labours under mania, of about five and a half years' duration. Having been removed from the asylum a short time ago, I can only give the result of her first examination. She confounded dark orange with scarlet; blues with various greys and lavender; and crimson, pinks and ruddy-browns.

CASE 7.—J. E—, æt. 37, labours under general paralysis, of nearly three years' duration. The appreciation of colour is almost destroyed in this woman. When admitted, she had a great variety of delusions about dress; she said she possessed a large number of silk dresses, some blue, some green, &c. It is now very questionable if she fully recognises any colour. She cannot arrange even the primary colours; occasionally she can pick up a yellow and give its name, but all others she appears to name by hap-hazard. She is not by any means so demented as many who have correctly arranged all the colours. She talks as fluently as ever of colours, but, so far as careful examination could make out, has almost entirely lost all power of perceiving them. To surrounding objects she gives most ridiculous shades, and appears quite unconscious of her remarkable errors.

CASE 8.—E. M. A. C—, æt 36, suffers from general paralysis, of about eight months' duration. She arranges correctly yellow, green, and brown. She cannot distinguish the difference between dark green, dark purple, and black. She arranges together light blues and purples, greys, and lavenders. With reds of all kinds she experiences great difficulty, and occasionally matches them with pinks and purples.

CASE 9.—F. H—, æt. 45, is an imbecile, with occasional attacks of excitement. A marked peculiarity in her case was that, whether she knew other colours or not, she only would look at and name pink. A most important fact, however, was that with pink she confounded light brown. Whether she presented other abnormalities of colour-vision it is impossible to say, as she absolutely refused to look at anything but what appeared to her pink.

The males examined numbered 302. Of these 39 suffered from epilepsy; 33 from general paralysis; 102 from dementia; 41 from chronic mania; 25 from melancholia; 25 from recurrent mania; 3 from mania; 3 from monomania; 1 from atrophy of the brain; 17 were idiots or imbeciles; and 13 were convalescent. Of the total number, 82 refused to answer, or were too demented or excited for their answers to be received. Correct information was received from 207, who attempted to arrange the colours, but 13 were more or less colour-blind : viz. 5 cases of dementia, 5 of general para-

lysis, and 3 of epilepsy.   Two of them—one a dement and the other a general paralytic—were almost insensible to colour; the others varied in degree, and in their leading features resembled the women whose cases have been re-corded.   It is therefore unnecessary to relate them in detail, as they shall all be re-examined, with many others, according to Goubert's method, and the results shall be recorded in next year's report.

In continuation of these introductory observations, it is my intention to examine other modifications of sight as observed in the insane.   Should circumstances prove favour-able, these shall be supplemented with ophthalmoscopic and microscopic observations of the retina, and by the record of enquiries made of convalescent patients as to the dis-turbances of vision which they experienced during their illness.

NITRITE OF AMYL IN EPILEPSY.

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A PAPER by Dr. Lauder Brunton, ' On the use of Nitrite of Amyl in Angina Pectoris,' which appeared in the ' Lancet ' of July 27th, 1867, first called my attention to the peculiar actions of that substance, which had been investigated by Guthrie, Richardson, and Gamgee. Impressed by the belief that it opened up a new path for the direction of treatment against various nervous disorders, I tried it in certain cases in which I hoped that benefit would accrue from its use; but, either because my experiments were not carefully designed, or because they were not perseveringly carried out, I obtained no satisfactory results. Disappointed, and hopeless of assistance from it in my special practice, I abandoned the enquiry, and did not employ nitrite of amyl again except as a palliative in angina pectoris and spasmodic asthma, until last year, when some observations on its physiological effects induced me once more to test its therapeutical properties. Being engaged in tracing out the areas of blushing, as induced by nitrite of amyl in different individuals and under different circumstances, with a view to elucidate the laws regulating the diffusion of that form of emotional expression, I was struck by the fact that the degree and extent to which the blushing caused by nitrite of amyl is manifested, are influenced by certain pathological states. I was particularly struck by the contrast which is presented by general

paralytic and epileptic patients, in their susceptibility, as regards blushing, to be acted upon by nitrite of amyl. I found that while the former may inhale a considerable amount without displaying any marked flushing, even of the face, the latter cannot breathe the smallest quantity without exhibiting extreme cutaneous hyperæmia over the face, chest, and neck. As the result of numerous observations, it became evident that general paralytic patients, and especially those in the advanced stages of the disease, are, with few exceptions, less amenable than epileptic patients to the action of nitrite of amyl. To illustrate this statement, the effects of the inhalation upon three patients of each class may be quoted.

1. Charles Edward P—, a general paralytic, in the third stage of the disease, altogether helpless and fatuous, was made to inhale 5 drops of nitrite of amyl. In fifty seconds his pulse, which had been 80, rose to 92; his breathing became hurried, and he manifested some restlessness; but no flushing appeared, although the inhalation was continued for two minutes. During a second inhalation of 10 drops, the only change noticed was violent grinding of the teeth.

2. Thomas D—, in the third stage of general paralysis, much demented, and very tottering in his gait, with a pulse at 90, was made to inhale 5 drops of nitrite of amyl. For two minutes no effects were discernible, then the pulse rose to 100, the breathing became hurried, and a slight degree of dusky flushing spread over the face; after a second inhalation of 10 drops, the same course of events followed; the blushing was trifling and transient.

3. Eli D—, in the second stage of general paralysis, labouring under multifarious delusions of an exalted nature, and considerable excitement, was caused to inhale 5 drops of nitrite of amyl. At the end of forty seconds his pulse sprung from 66 to 90, and well-marked flushing of the face appeared, the breathing at the same time becoming laboured. Subsequently, a few scattered red blotches were seen—over the front of the chest, and in the centre of the back.

1. George O—, an epileptic, who suffers from frequent fits, and is much demented, was made to inhale 5 drops of nitrite of amyl; his pulse being then 62, and his breathing quiet and regular. In ten seconds his respiration was perceptibly deepened; in twenty seconds a deep red blush came out all over the face, forehead, and ears, and spread on to the front of the chest, over the sternum, very rapidly; this blush spread over the neck—where, however, it was less intense in colour, and where violent throbbing of the carotid arteries could be seen—and then over the shoulders, with the exception of a triangular patch of skin over each detoid muscle, which was left of its ordinary white colour. From the shoulders it ran down the dorsal aspect of the arms as low as the wrists, being very vivid down to the elbows. It also spread

down the middle of the back between the scapulæ, as low as the 10th dorsal vertebra, in a well-defined band 3 inches in breadth. From the chest it extended on to the abdomen, to within a couple of inches of the umbilicus. It appeared again on the crest of each ilium, and extended over the hips and outer aspect of both thighs half way to the knees. The blush was everywhere very bright, and uniformly diffused. With it came greatly-increased mental liveliness. While the blush remained for about three minutes, and for some time afterwards, George O— was talkative, and more intelligent then than it is his wont to be. A second inhalation of 10 drops, a quarter of an hour after the first, produced the same effects, but after a longer interval and to a less extent.

2. George Richard L—, who has been subject to epileptic fits for many years, and who had recently emerged from an attack of excitement, was made to inhale 5 drops of nitrite of amyl, his pulse being then 84 and feeble, and his complexion dusky and florid. In thirty seconds his breathing grew hurried, while his pulse rose to 120; his pupils began to oscillate in a remarkable manner, and continued to do so throughout the inhalation; his eyeballs being at the same time exceedingly restless. In forty seconds deep red flushing of the face and head occurred, shading down the neck, into a diffused reddish blush over the chest, and as low as the waist, and extending along the pectoral muscles on to the shoulders, and thence down the dorsal aspects of the arms and forearms. A diffused blush was also visible over the back, most marked over the supra-spinous fossæ of the scapulæ, and down the vertebral column to the lumbar region. There was also a long, very distinct, isolated blotch of blushing over the crest of each ilium. During the inhalation there was no motor disturbance, but mental activity was stimulated. After a second inhalation, ten minutes later, the pulse again rose, and the pupils oscillated as before. The blushing was more distinct, and, further, a few scattered patches of blushing were seen on the flexor aspects of the arms, while a bright red diffused blush spread from the iliac blotches previously mentioned, over the hips and buttocks, and down the outer and posterior surfaces of the thighs, as low as the knees.

3. William E—, a chronic epileptic, with a dusky complexion, and pulse at 72, was made to inhale 5 drops of nitrite of amyl. In thirty seconds his pulse went up to the rate of 126 per minute, and brilliant blushing of the face, forehead and ears appeared. In forty seconds the neck was flushed, and the carotids were pulsating violently. In forty-five seconds a diffused red blush had pervaded the whole of the front of the chest, and the abdomen as low as the umbilicus, and had spread along the surface over the pectoral muscles on to the shoulders, and thence down the back of the arms and forearms. There was also a diffused blush covering the whole back. Below the umbilicus there was a belt of white skin, and then came another area of blushing, extending from the iliac crests over the buttocks and posterior surface of the thighs. During the whole of the inhalation, and for some time afterwards, the patient indulged in immoderate laughter, and was unusually lively. During a second inhalation of 5 drops, the effects were precisely as noted above.

These cases exemplify in a striking manner the comparatively high susceptibility of sufferers from epilepsy to the action of the nitrite of amyl; and many others might have been adduced to establish the same fact. In almost all the epileptics who were made the subjects of experiments, the blushing brought on by the nitrite of amyl was widespread and intense, while other indications demonstrated that the effects of this agent were not confined to the cutaneous capillaries. Respiration was invariably quickened, the pulse was accelerated, and vascular tension diminished, and at the same time the mental powers were stimulated. In a few instances a single muscle, or group of muscles, was thrown into movement, but, as a general rule, muscular contractility seemed to be reduced during the inhalation. A fit was never in any case brought on by the inhalation, although that was frequently administered in the midst of a periodical series of fits ; nor did a seizure in any instance follow it in so short a time as to suggest that it might have been instrumental in hastening its advent.

Reflecting upon these phenomena, and especially on the high susceptibility of epileptics to the action of nitrite of amyl, I was led to enquire whether a substance so powerful in its operation upon those afflicted by this particular disease might not be made useful in controlling the course of the malady. It is clear that epilepsy is dependent upon ' an unstable condition of nerve tissue in some portion of the nervous system permitting occasional discharges,' but it is clear also that that unstable condition, and those discharges, are often dependent upon more remote causes, and that among these, changes in the circulation and vascular system hold a prominent place. No true epileptic fit occurs without affording visible proofs that vascular changes are involved either in its evolution or its consequences. Pallor, redness, or duskiness of the countenance, throbbing of the arteries, palpitation of the heart, distension of the veins, and lividity of the surface, are recognised as being almost as essential to genuine epilepsy as unconsciousness and clonic spasms. One at least of these indications of vascular or vaso-motor

derangement is present in every case ; and in a large propor-
tion of cases, one or more of these indications hold such a
relation to the other constituent elements of the fit, as to
suggest that vascular disorder is a necessary condition of
its development.   Among the prodromata of the epileptic
paroxysms, palpitation of the heart, and violent pulsations
of arteries, swelling and blueness of the hands, and heat,
rigors, and sensations of cold, must be enumerated.   Then
among the phenomena of the fit itself, pallor of the face
is perhaps the earliest and most constant.   In some cases,
indeed—those classified under *petit mal*—this pallor is the
only outward sign of the fit, and corresponds with the mo-
mentary unconsciousness or loss of perception and volition
in which it consists.   A deadly whiteness pervades the face,
there is a moment of mental vacancy or of vertigo, then a
little confusion of thought, and the transient attack, the
identity of which with epilepsy is shown by its leading up to
and alternating with unmistakable convulsive seizures, is
over.   In epilepsy of a severe nature, characterised by tonic
and clonic spasm, pallor of the face, to a perceptible extent,
is very frequently the harbinger of each attack.   A ghastly
whiteness of the countenance precedes any muscular
rigidity or twitching, or any impediment in the respiration
or dilatation of the pupils.   And even when the contraction
of the capillaries of the face is not noticed, it is sometimes
present, although it escapes detection owing to darkness of
the complexion, or the shortness of its duration.   There can
be no doubt, however, that it occurs to a recognisable degree
in a very large number of cases.   Delasiauve constantly
observed it at the commencement of epileptic attacks, and
Dr. Russell Reynolds says of it that it occurs ' immediately
before and at the very outset of the attack in many ; it is not
present in all ; and it occurs more certainly and more
notably in females than in males.'   Now, in the severer, as
well as in the milder kinds of epilepsy, this loss of colour in
the face coincides with the loss of consciousness.   At the
same moment that the superficial vessels contract, those
within the cranium contract also, by virtue of a command
from their common centre, and then results a state of cere-

bral anæmia which means, we know, an interruption of mental activity, and then convulsions. I should be disposed to lay it down as an inflexible rule, that in all cases of epilepsy in which loss of consciousness occurs at the commencement of the fit, there is an arrest of the blood supply to the brain in consequence of spasmodic contraction of the intra-cranial vessels. Indeed, it is difficult to understand how loss of consciousness at this period could be otherwise produced. The discharging lesion and the process of discharge itself do not cause unconsciousness. Partial convulsions, affecting one limb or set of muscles, occur frequently without any obscuration of the mind, and even general convulsions are sometimes witnessed throughout which consciousness is perfectly retained. Some epileptics have fits, in which tonic and clonic spasms of the face, hands, and arms, anticipate by several seconds, or even minutes, any impairment of perception or intelligence ; they are aware of all that is going on around them, can answer questions, and can recall, when the fit is over, what took place during the continuance of these spasms. A few can even recall the cry which they emitted at the moment when the seizure was fully developed, and they dropped into unconsciousness. It is clear, then, that the loss of consciousness at the commencement of a fit is not dependent upon the mere process of discharge, and it is equally clear that it cannot be traced to venous congestion of the brain or insufficient decarbonisation of the blood. These conditions are due to spasms of the muscles of the neck, and perhaps also of the cerebral arteries and capillaries, and to defective respiration, owing to closure of the glottis, or fixation of the thoracic walls and diaphragm, and cannot, therefore, be established until the tonic spasm has been in operation. Besides, the features at this point are not at all significant of asphyxia. To what, then, can the initial unconsciousness of epilepsy be ascribed, save to arrest of the cerebral circulation, owing to contraction of vessels ; and to what can this be ascribed, save to some irritation propagated from their vaso-motor centre. No reasonable doubt can be entertained that cerebral anæmia thus induced is the explanation of the primary unconscious-

ness in epilepsy, and that wherever the unconsciousness is primary, such cerebral anæmia is present, whether or not it is signalised outwardly by pallor of the face.

How far the contraction of the vessels and cerebral anæmia, which inaugurate so many epileptic fits, are directly responsible for the subsequent steps and stages of the seizure remains to be determined. The experiments of Sir Astley Cooper and of Kussmaul and Tenner, make it certain that they are adequate to the induction of convulsions, but whether the convulsions in epilepsy are set in motion by them, or by the irritation out of which they themselves arise, cannot as yet be definitely settled. But, however that may be, contraction of vessels and cerebral anæmia are evidently important elements in the epileptic state. Withdraw them, and the morbid product is broken up and decomposed. Prevent them, and a fit of the ordinary type seems impossible. Now, the nitrite of amyl indisputably prevents contraction of vessels and cerebral anæmia during its inhalation. It is exceedingly potent in epileptics, and acts with great force upon those vessels which in them seem to be in a state of preternatural excitability or weakness. Might it not be useful in counteracting that spasm which is the starting point of so much that is evil in epilepsy?

Arguing in this way, I was led once more to try the nitrite of amyl in epilepsy. A previous trial had resulted in failure, and Dr. Lauder Brunton also had found no benefit derived from it in a case in which he had employed it; but a point of great moment not previously attended to was now judged to be the precise period of administration. It seemed that if the nitrite of amyl could be given as an inhalation immediately before the fit, the spasm of vessels might be prevented, and so the whole sequence of morbid events averted. And a fit averted in epilepsy is no slight gain ; it is, in fact, a step made towards recovery, and a postponement of those degenerative consequences which are as a rule developed in proportion to the frequency and severity of the fits. To interrupt a pathological habit is to give a chance of recovery ; to control the fits is to limit the destructiveness of epilepsy.

In order to secure the administration of the nitrite of

amyl immediately before the fit, it was requisite to find a
case in which warning of its approach was given.   But, un-
fortunately, in lunatic asylum practice an aura is of rare
occurrence in epilepsy.   Using the word in its widest sense,
as including not merely the peculiar sensation as of a wind
or vapour beginning at some part of the periphery and
travelling towards the nervous centre, to which it is more
strictly applicable, but all prodromata, all symptoms, psy-
chical, sensory or motor, which immediately precede a parox-
ysm, an aura is rare in a lunatic hospital.   It is so, firstly,
because the epileptics who are received into such an hospital
are always far advanced in the disease; in them the aura, if
it ever existed, has been merged in the fit, as every aura
tends at last to be; and, secondly, because their feelings and
intelligence are blunted, so that they do not recognise nor
describe any aura which they may experience.   Those of
them who have their fits in groups either at periodical or
irregular intervals, become dull and lethargic, or irritable
and violent, when the fits approach, and continue so until
some time after their cessation.   They are seen sitting, sullen
and heavy, or wandering about restless and turbulent, and
the nurse says, 'they are having their fits,' and deems it
a sufficient explanation.   But even with them there is no
distinct premonition of each individual fit, and the most
experienced observer may be at fault in predicting when one
is going to happen.   Dr. Russell Reynolds, speaking of epi-
leptics in the general community, says that prodromata are
in some form or other more frequently positively present than
positively absent.   Their presence was recorded in 43·2 per
cent., their absence in 40·7 per cent. of the cases which he
tabulated.   Dr. Sieveking states that out of 58 cases of which
he had preserved careful notes, 30 showed some indications of
the approaching paroxysm; and Romberg found premonitory
symptoms in one half of his epileptic patients.   With the
epileptic inmates of a lunatic asylum the reverse holds good.
Trustworthy prodromata of any kind are exceedingly un-
common.

   When first desiring to test the efficacy of nitrite of amyl
in warding off a fit, at the very impingement of which it

was administered, I could find no case in which it was possible to introduce an inhalation between an aura and the paroxysm. Either the one followed the other with such rapidity, or both were so uncertain in their occurrence, or the condition of the patient was such, that no reliable experiment was possible. Under these circumstances, I resolved, as the next best test within my reach, to administer the nitrite of amyl regularly to a patient who was at that time having one fit every day with considerable punctuality. I hoped that the nitrite, when inhaled near the time at which the fit was due, might so dilate the vessels as to oppose any tendency to their contraction, or might so affect the vaso-motor centre as to alter its condition, and dissuade it from issuing a spasmodic fiat. Whether my theory was good or bad, I was not disappointed in the results which I had ventured to hope for ; indeed, I obtained results more favourable than my most sanguine expectations. These will be best set forth by a brief report of the case.

Eliza W—, 27 years of age, single, worsted-reelor, from Bradford, was admitted October 4th, 1872, being brought from the Parkside Asylum near Macclesfield, where she had been under treatment for three months. On the admission form, which is not, however, altogether trustworthy, it was stated that she had become epileptic only eight months before her removal to the Parkside Asylum, and that no cause for the fits by which she is afflicted could be designated. According to her own statement, she had had no fits for two months before her removal here, but on the fourth day after her reception a fit occurred. She turned pale, screamed, and then fell. No genuine convulsions were noticed by the nurse, who stood by. She picked her clothes with her fingers in a singular way, especially with the fingers of the left hand, remained lying insensible on the floor for five minutes, and then arose as if nothing had happened. For two days after the fit she refused her food, imagining it was poisoned, and then came another fit. After that fits were frequent, and were accompanied by intense mental irritability. She was perpetually quarrelling with her companions, and making unfounded charges against them ; sometimes she tore clothing, and destroyed furniture, merely to vex the nurses who had charge of her. The fits came suddenly, without warning, but invariably with death-like pallor of the face, which was exceedingly obvious in her, as her complexion is naturally florid. From January 1st, 1873, to March 26th, a period of eighty-five days, she had eighty fits, one each day, with great regularity. Occasionally two days passed without a fit, and then generally two fits occurred on the third day. During the month of March she had one fit each day, at no fixed hour, but most commonly in the forenoon. On March 27th, nitrite of amyl inhalations were commenced, 5 drops being administered three times

a day. The fits were immediately and abruptly discontinued. No fit occurred from March 27th till April 15th. The inhalations always produced well-marked flushing of the face, ears, and neck, with deepened respiration, acceleration of the pulse, which bounded from 70 to 120, and a peculiar quivering of the fibres of the orbicularis palpebrarum muscle, on the left side, and of the depressor anguli oris on the right side. The patient enjoyed the effects of the inhalations, which made her, she said, feel warm and comfortable. When the inhalation was over, the flushing disappeared rapidly, and the pulse fell at once, and without any intermediate stages of slackening, from 120 to 80. Whenever the fits ceased to assail her an agreeable change took place in the disposition and temper of Elizabeth W—. She was no longer irascible and troublesome, but amiable and useful. On April 15th a fit of the usual character occurred, and after that occasional fits occurred; the inhalations being only irregularly administered. Up to the present time, however—and no inhalations have been administered lately—the fits are comparatively infrequent, and the patient's mental state is altogether more satisfactory than it was six months ago.

This case was so encouraging that I sought further opportunities of putting the nitrite of amyl to the test, with reference to its power of averting an impending fit, and had shortly the advantage of doing so in a crucial and conclusive manner.

In a rabbit rendered artificially epileptic by Professor Ferrier, a severe fit followed exactly ten seconds after the momentary application of the electrodes to the right hemisphere (the fit was characterised by drawing of the head to the left, violent convulsive movements of the left lip, eyelid, and left fore paw). This experiment was repeated twice, and then, in a third experiment, at the same instant that the electrode was applied to the brain, a piece of lint, upon which ten minims of nitrite of amyl had been dropped, was placed over the mouth and nostrils. After this application the animal continued to breathe naturally, and did not close its nostril or stop its heart, as rabbits, to which chloroform is administered, so generally do. No fit nor muscular twitching of any kind occurred. After an interval of five minutes the electrodes were again applied to the brain, nitrite of amyl being at the same time inhaled, and with a like result. No perceptible effect followed. After another five minutes, the electrodes, the secondary spiral being at 3 centimètres, were again applied, while nitrite of amyl was again inhaled, and this time, after 40 seconds, a peculiar munching movement of the

upper lip with slight drawing of the head to the left was observed. After another five minutes the electrodes, the secondary spiral again at 3 centimètres, were again applied, and were kept applied for ten seconds. At the end of thirty seconds, twitchings in the upper lip commenced, after which the head was strongly drawn to the left, and a distinct fit occurred, as severe as those which occurred before the first nitrite of amyl inhalation.

In another rabbit which I have since subjected to experiment very similar results were obtained. When the irritation applied to the brain was slight and only instantaneous in operation, the fit which invariably followed such irritation, when no interference took place, was arrested by the inhalation of nitrite of amyl. When the irritation was more severe and continuous, the fit, if not averted, was postponed for a certain number of seconds, and was generally milder in character.

Now here were fits arrested or mitigated by inhalations of nitrite of amyl immediately prior to a fit, between the application to the surface of the brain of an irritant, which may be regarded as closely analogous to an aura, originating in the peripheral expansion of a nerve, and the discharge of nerve energy to which that irritant gave rise. No change in the vascularity of the brain was visible to the unaided eye, but there could be little doubt that the nitrite of amyl acted upon the rabbit as upon the human subject, by increasing the calibre of vessels. An opportunity occurred shortly afterwards of satisfying myself, that that substance, when inhaled at the proper time, may avert a fit, in the human subject who is suffering from epilepsy, as surely as in the rabbit which has been made epileptic.

On Sunday, May 18th, 1873, Elizabeth W——, aged 25, who had been epileptic for eighteen years, and an inmate of the West Riding Asylum for five years gave warning of the approach of a fit in her usual manner, by nodding her head continuously and monotonously, and by sobbing piteously now and again. This was at 2 p.m., and the nurse, taught by experience, knew that convulsions and unconsciousness would supervene in about two hours from that time. An inhalation of 7 drops of nitrite of amyl was at once prescribed, and the effect of this was, that the nodding and sobbing ceased, and that the patient grew quiet, and more lively and intelligent than it is her wont to be. At the end of two hours, 5 drops of the nitrite of amyl

were again administered, and no fit occurred. At 6 and 8 P.M., inhalations again took place. The patient remained free from fits, and passed a good night. On the following day, the inhalations were omitted. At 4 P.M., the nodding of the head and sobbing again commenced ; and now, in order that the correctness of the nurse's observation might be tested, no interference was permitted, a strict watch being maintained. At 6 P.M. precisely, a severe fit, characterised by general convulsions, and prolonged stupor occurred. Since then, another fit, premonished in the usual way, has been altogether warded off, by periodical inhalations at intervals of every . two hours for two days.

With the purpose of further testing the nitrite of amyl, the night attendants, who are in charge of certain dormitories in which epileptics sleep, and who watch them constantly to prevent any cases of suffocation by rolling on to the face during a fit, were provided with bottles containing a little of it, and were instructed to employ it whenever they knew with absolute certainty in any case that a fit was beginning, noting in their report books the effects of the inhalations. One of these attendants has had occasion to employ it twice, and his reports in his own words are as follows :—' 21st April, 1873. I applied the nitrite of amyl to Michael D— just as I observed a fit coming on him, and the effect was, his breathing became heavy for about half a minute, without any convulsions, and had just the appearance of a man fainting.' ' 22nd April, 1873. I applied the nitrite of amyl to Edward B— this morning, at 3·45, whilst a fit was coming on; the effect was much better than in Michael D—, as he came to in about twenty seconds.' On asking the night attendant to explain these entries more fully, he informed me that Michael D— had started up in bed suddenly, as he always did before a fit, with his head turned to one side, and his eyes staring fixedly at the moment when the inhalation was administered, and that he (the night attendant) had no doubt whatever that a fit was imminent, and was greatly surprised when the patient subsided upon his pillow, and lay in what he termed a half-fainting state, without the slightest motor agitation. As regards Edward B—, he informed me that the fit had actually commenced in its ordinary way with rigid stretching of the hands by the side and turning up of the eye-balls, when the nitrite was

held over the mouth and nostrils. Complete recovery took place in twenty seconds. The other night attendant succeeded in trying the nitrite of amyl in one case only, that of Thomas S—, respecting which he wrote this report: ' May 29th, 1873. Thomas S—, at $3\frac{1}{2}$ A.M., showed symptoms of a fit, he inhaled 5 drops, it passed away, but he appeared rather stupid for forty seconds.' This report was supplemented by the statement made to me that the symptoms of the coming fit consisted in stiffening of the arms, and fixing of the eyes, which long experience had taught the night attendant to recognise in this man as infallible warnings of a coming fit. Never before had these symptoms been observed without a fit following in the course of a few seconds. In this case, as in those reported by the first night attendant, the inhalation could not be said to intervene between an aura and the time for the occurrence of a fit; it really corresponded with the introduction of the fit itself, the period of tonic spasm, and yet it was effectual in bringing the seizure to an abrupt and unusual termination. The stupidity, or fainting, as it was called, may, I think, be looked upon as analogous to cerebral surprise or shock after a slight concussion. No doubt in these cases spasm of vessels had already taken place to a certain extent, or in certain regions. In the case of a girl, Harriet H—, who screams automatically for a few minutes before she is prostrated by a fit, the prevention of the fit by a nitrite of amyl inhalation is not followed by any trace of mental dulness or syncope.

The nitrite of amyl has now been used to ward off fits in several other cases, upon the details of which I shall not here enter. The results of all my experiments is to convince me that it will be found invaluable in many cases, in not only postponing but altogether preventing epileptic seizures. The utility of an agent possessing this power can scarcely be exaggerated. It will, I believe, supersede other methods of attempting to avert the fit by acting upon indications afforded by the aura. Pressure upon or ligature of a limb, section of a nerve trunk, or cauterisation of the surface from which an aura originates, have done good service, in certain cases, in hindering the accession of seizures, but the nitrite

of amyl appears to be a more ready and certain means for compassing the same end. A viñaigrette or small stoppered bottle, containing a sponge soaked in nitrite of amyl, and carried in the pocket so as to be at hand, on the occurrence of an aura, will, I think, be found a safeguard to many sufferers from epilepsy. Wherever there is time, after the initiation of the aura and before the development of the proper phenomena of the fit, to breathe the nitrite of amyl freely, the fit with its terrible accompaniments and disastrous sequelæ may in many instances be not merely postponed, but abolished. Of course, I am aware that it is only in certain kinds of epilepsy that this treatment will be available. Epilepsy is a generic term, and it includes many species and varieties. Our object must be to differentiate these, and to decide in which of them the nitrite of amyl may be resorted to with most benefit. In the meantime it is clear that its use is contra-indicated in certain classes of convulsions sometimes confounded with epilepsy. It might do serious mischief when convulsions are due to uræmia, to clot, to disease of the cerebral vessels, or to the immediate effects of injury of the head. Under such circumstances, however, auræ are rarely if ever met with.

But there is another epoch in epilepsy besides the pause between the aura and the fit, when, according to my experience, nitrite of amyl may prove beneficial, and that is at an advanced stage, when that alarming condition called the *status epilepticus* occurs. That condition consists essentially in a succession of fits linked together by intervening unconsciousness. At first there has occurred one fit in all respects similar to those from which the patient usually suffers, with or without premonitory symptoms, the convulsive movements lasting for a period rarely exceeding three minutes, and being followed by coma or comatose sleep. But before this coma or comatose sleep has resolved itself into bewildered wakefulness, another fit happens, and after that a third, and so on for hours and even days together. When the *status* is at its height, the convulsive seizures succeed each other with extraordinary rapidity, so that the limbs are scarcely laid at rest after one fit before they are

tossed and contorted by another. And even in the intervals which separate the fits, frequent muscular twitchings testify to the urgency of these motor impulses, which every now and then swell into explosive activity. The heart's action is rapid and tumultuous, and the respiration is quick and shallow, or slow and laboured. The temperature is considerably raised, and the surface is bathed in profuse perspiration, while the features are swollen and livid and the lips purple. The general state of the patient betokens, 1st, exhaustion, cerebral and systemic, from the number of fits; 2nd, coma, from venous congestion of the brain; and, 3rd, asphyxia, from closure of the glottis, or prolonged and repeated fixing of the chest-walls, with gradual congestion of the lungs.[1]

Now the *status epilepticus* is a most dangerous complication of epilepsy. It is often the last and fatal phase of the disease, and its treatment has always been a matter of anxiety and doubt. The bromide of potassium, the head and front of all systems of treatment of epilepsy in recent years, is worse than useless during the *status*, either alone or combined with sumbul, ammonia or chloroform. Bleeding in plethoric subjects to a small amount has afforded relief, but only of a temporary nature, and, of course, in the majority of epileptics, it could not for a moment be entertained as a possible expedient. Atropine is unavailing, as the *status* is sometimes developed in those patients who are distinctly under its influence. Opium in any form is simply fatal. Ergot or ergotine injections are not trustworthy. Stimulants, though indispensable, are not all-sufficient; and ice to the spine, although very valuable, cannot be pronounced curative. Deploring the powerlessness of the means hitherto in use to control the *status epilepticus*, and seeking after some more certain method of treatment, I made a careful analysis of the condition as presented to me in several cases, and

[1] Perhaps some chemical change in the brain is also operative in the *status epilepticus*. In three cases in which death had resulted from it, and in which examinations were made in the post-mortem theatre of this Asylum, a strong odour of chloroform pervaded the room whenever the head was opened. The odour was most distinctive, and was at once recognised by everyone present. In one of these cases the spiritus chloroformyli had been administered during life, but in the other two chloroform in any form had not been partaken of.

came to the conclusion that a slow or partial asphyxia was in great measure responsible for its phenomena and fatality. It seemed to me that the *status* supervened when any intense eccentric irritation, such as a wound of a nerve, a loaded state of the stomach or bowels, or a uterine or ovarian de- rangement so stimulated the seat of the discharging lesion in a confirmed epileptic, as to entail two or three successive discharges with protracted fixation of the chest in each. This fixation necessarily causes a retardation of the blood current in the pulmonary capillaries, and imperfect aeration of the blood. This imperfect aeration creates a difficulty in the passage of the blood through the systemic capillaries ; the arteries, and left ventricle are distended, and the heart works heroically to overcome the obstacles in the way of the circulation. But the brain and nerve centres are speedily affected by the bad supply of bad blood, and give vent to their dissatisfaction in renewed convulsions, which again interfere with respiration and deteriorate the blood. What would seem an endless chain of morbid activity is thus set in motion. But the chain is not endless. The lungs, in which the interchange of carbonic acid and oxygen cannot be carried on, cease to transmit even venous blood, and become engorged, and the right side of the heart and veins are loaded with dark blood which poisons the tissues ; the left side of the heart and arteries contain only a little venous blood which cannot support the functions of the muscular or nervous systems, so the brain and heart become paralysed, and death ensues.

Supposing that this is a correct explanation of the *status epilepticus,* the problem in treating it evidently is to find some remedy which will either reduce the excitability of the dis- charging centre, or relieve the asphyxia. In deliberating upon the above proposition, it occurred to me that the nitrite of amyl might fulfil at least the latter indication. Perhaps it might diminish the excitability of the nerve centres, almost certainly it would relieve the asphyxia. Guthrie had pro- posed it as a resuscitative in drowning suffocation and pro- tracted fainting ; and there seemed every reason to anticipate that it would relieve the asphyxia of the *status epilepticus.*

By stimulating the respiratory movements and dilating the pulmonary-capillaries it would favour the onward flow of the blood, and promote its oxygenation. By dilating the systemic capillaries and lessening arterial tension, it would relieve the left side of the heart, and so aid in the restoration of its rhythmic power. By gradually re-establishing the circulation and the propulsion of better blood through the brain and muscles, it would conduce to the return of consciousness. At any rate, it seemed well worthy of a trial, and accordingly in the next attack of *status epilepticus* that happened in this asylum it was had recourse to. Since then it has been used in nine cases. Of the ten cases in all in which it has been employed, eight have terminated in recovery, two in death. With regard to the fatal cases, in one it seemed simply to fail to afford relief, and I can offer no explanation of its failure, unless the fact that the patient had been epileptic for nineteen years, and was in a condition of stupor and exhaustion when the *status* came on be regarded as an explanation; in the other it had certainly not a fair trial, as the patient had had sixty fits in the space of three hours, and was actually at the point of death when the first inhalation was administered. Even in the fatal cases, however, one or two instructive and encouraging points were noted. In John S——, the breathing became deeper and fuller for a time after each inhalation, and in Amelia K——, a change in the character of the fits was induced. The convulsive movements, which recurred every five minutes, always commenced in the eyes, which were turned upwards and oscillated violently. After a few seconds, twitchings begun in the hands and spread up the arms. Next the legs were affected. Several times as the nitrite of amyl was inhaled as a fit was commencing, the convulsive movements were confined to the eyeballs; these were turned upwards and oscillated as usual, but the arms and legs did not become involved.

Turning to the successful cases, I need only say before briefly describing a few of them, that they leave no doubt whatever on my mind that nitrite of amyl is an invaluable remedy in the *status epilepticus*. But for that agent several of them must have terminated in death, and the

rest must have been greatly protracted. Having unfortunately had a large experience of the *status epilepticus*, I can speak with some confidence as to the merits of the various systems of treatment that have been recommended for that hazardous condition, and I have no hesitation in saying that the nitrite of amyl inspires me with new hopefulness in encountering it. Cases which I should formerly have despaired of I shall now regard with less apprehension. Carefully attending to the general condition of the patient, seeing that he is placed in a warm and well ventilated room, that he receives a liberal supply of nutriment, that he is kept quiet, that his position is frequently changed, that cleanliness is observed, and that stimulants are administered by the mouth or rectum according to the indications present, I shall in future trust to nitrite of amyl as the most efficacious remedy at my command. Of course other auxiliary remedies are not to be ignored. Occasionally an emetic or an aperient injection may contribute to recovery. When robust, full-blooded subjects have to be dealt with, a small bleeding may work wonders. I have seen patients of this class who were in a state of profound coma, so that powerful stimuli induced no reflex reaction, suddenly raised into sensibility and obscure consciousness by the withdrawal of a few ounces of blood. Then ice to the neck and spine sometimes does unmistakable good, while sinapisms to the legs and feet are beneficial. On the whole, however, nitrite of amyl operates with a directness and certainty such as I have never witnessed in connection with any other remedy in the *status epilepticus*.

Ellen C—, aged 43, a mill-hand from Huddersfield, was admitted on November 25, 1871. After the birth of a child, eight years previous to her reception here, she became epileptic, and has suffered from fits ever since. These at first recurred every month, but latterly they have been more frequent, and have been accompanied by what is called 'raging madness,' necessitating the use of strong ropes to tie her down in bed. Being altogether unmanageable in the workhouse, she was sent here for safe-custody. During the earlier part of her residence here, and under bromide of potassium treatment, she improved in general health, and the fits were less numerous. She remained, however, childish in intellect and in manner, and impetuous in temper, violently resenting any opposition to her wishes. On January 22nd of the present year, being still under the bromide of potassium treatment, and having had no fits for two months, she complained

of rigors, which proved to be the starting point of a sharp pneumonia of the left lung. On the sixth day of the pneumonia, her temperature rose 104·2° F., her pulse to 124, and her respirations to 44 per minute, but after that she began to improve, and on the tenth day was pronounced convalescent. Although carefully nourished and stimulated throughout her illness, she was left greatly exhausted by it. The bromide of potassium had been omitted on the occurrence of the rigors. No fits happened during the pneumonia, but on February 4th when convalescent, she had one of a rather severe character. On the 6th she had two fits, on the 7th three, and on the 8th fifteen. At this point the *status epilepticus* was established, as consciousness was not fully recovered between the paroxysms, and as the breathing became laboured, and the heart's action excited. Bromide of potassium in 40 grain doses every four hours was prescribed, with 6 ozs. of port wine per diem. On the 9th she had eighteen fits, and was continuously unconscious. Sinapisms were applied to the legs and feet, and nourishing injections were administered. On the 10th she had twenty-five fits, one every hour, with tolerable punctuality, and between the fits, which were terribly severe, muscular twitchings were noted. Her pulse was 84, and feeble; her breathing was hurried, and her temperature was 102·6° F. in the morning, and 103·2° F. in the evening. Her face was of a livid purple colour. The bromide of potassium was ordered to be discontinued, and ice applied to the spine. On the 11th she had a constant succession of fits, and was deeply comatose, so that she could not be roused by the most powerful stimulation. Reflex action was abolished, and tickling or pricking the soles of the feet occasioned no movement. The temperature in the morning was 102·1° F., and in the evening 104·0° F. The pulse was 130, the whole body was bathed in perspiration, the surface was dusky. Two ergotine injections, 10 grains each, were administered hypodermically during the day, and the application of ice to the spine was continued, but no amelioration of the symptoms took place, and the patient, who had had ninety-one fits in the previous twenty-four hours, appeared to be sinking rapidly at 9·45 P.M., when the first nitrite of amyl inhalation was administered. The immediate effect of the inhalation was a slight degree of rousing; the patient moaned and moved her head on the pillow; in ten minutes a second inhalation of ten drops was administered, when some dusky flushing of the face was observed, and more decided uneasiness, expressed by moaning and movements of the head. During the night there were only six fits, and the next morning, when the inhalations were resumed, distinct improvement had taken place. The pulse was 100 and stronger, and the temperature 100·0° F. The skin had more of its natural colour, and reflex action was re-established—tickling the soles was at once followed by movements of the legs. From that time no more fits occurred, consciousness slowly returned, and gradually, recovery took place, retarded however by a bed sore, and phegmasia dolens affecting both legs. The patient is now considerably better than she was a year ago.

Lydia H—, aged 26, single, a factory-hand, from Leeds, was admitted on August 8, 1868, having been then epileptic for many years. She was in a state of dementia, in which she has continued ever since, being occasionally liable to outbreaks of maniacal excitement. She was frequently afflicted by fits of a severe description. On February 10th, she had during the evening,

from 8 to 10 P.M., ten fits of great severity in rapid succession, and without recovery of consciousness. During the night there were no fits, but on the following morning four occurred, and after them came a brief paroxysm of excitement. On the 12th, 13th, and 14th, she had one fit each day, and on the 15th passed suddenly into the *status epilepticus*, having eighteen fits that day. Brandy was prescribed, but without effect, as on the following day she had twenty-one fits, and was deeply comatose. Ergotine injected hypodermically, ten grains at a time, was next had recourse to, but that failed to produce any favourable change in her condition. On the 17th she had twenty-four fits, and ice bags were then applied to the spine, but they too were without effect. On the 18th she had forty fits, and on the 19th up to 1 P.M. thirty-four. At that time, when she appeared quite worn out and at the point of death, her pulse being 130, her temperature 103·0° F., just after a fit, the first inhalation of nitrite of amyl was administered. An unexpected lull at once followed, no fits occurred for two hours, two more inhalations having been meanwhile administered; then came one fit. Up till 12 midnight only four fits occurred, and subsequent to that none until the 20th, two days afterwards, when another ordinary fit occurred. Consciousness was rapidly restored, while all traces of the *status* vanished in a few days.

John W——, 50 years of age, married, an iron-sheet roller, from Barnsley, was admitted on November 20, 1872. He was then labouring under violent excitement, the result of epileptic fits, of which the first occurred in August, 1872. These fits, which generally came on when he was at work, were supposed to be in some way dependent upon the great heat to which he was exposed when employed at his trade. It was thought also that a predisposition to them might have been established by the intemperance in which he had indulged, until within twelve months of their incursion. No direct cranial injury was known to have been incurred by him, but some years ago his right eye was destroyed by a flash from a roller, which wounded the sclerotic, and resulted in inflammation and disorganization of the eyeball, 'and adhesion of the lower eyelid. Soon after his admission here the excitement subsided, leaving behind it, however, considerable fatuity, a staggering gait, and some thickness of speech. No fits occurred until December 25th, when a series of convulsive seizures, always commencing on the left side, and then spreading to the right side, and following each other with considerable rapidity for a period of twelve hours, prostrated his strength, and left him more stupid than before. Subsequently to that, groups of fits occurred at tolerably wide intervals, and without any perceptibly disastrous effects until May 6th 1873, when a terrible epileptic outburst happened. On that day he had sixteen fits, all severe and long-continued. On the following day, May 7th, he had twelve fits of a like description, and on May 8th he had sixteen. Stimulants and bromide of potassium, which were given copiously, seemed to have no effect; he did not recover consciousness between the fits, and only swallowed with difficulty. On May 9th he had sixteen fits, and on the 10th, thirteen. On the 11th his condition was most critical. He had thirteen fits up till 2 P.M. He lay on his back deeply unconscious, breathing stertorously, with livid purple features, and streaming with perspiration, which stood in beads on his forehead, and saturated the bed-clothes around him. His pulse was 140, and his tempera-

ture in the axilla 103·0° F. Nitrite of amyl was now resorted to, the bromide of potassium being discontinued. Five drops were given him to inhale every hour. Improvement was inaugurated after the first inhalation; his breathing became less laboured. During the remainder of the day, from 2 P.M. till 12 midnight, there were only three fits, and these were not so protracted as previous fits had been. Throughout May 12th there were only five fits and gleams of consciousness intervened between these, while the pulse fell to 120, the temperature to 100° F., and swallowing was more easily accomplished. On May 13th there were only three fits, and on the 14th two. The patient was now quite conscious, and could answer questions. The inhalation was given once in three hours. On the 16th he had one fit, and on the 17th none. After that he passed into his ordinary state of bodily and mental health, and was able to take part in domestic work. So severe a case of the *status epilepticus* as his I never saw terminate in recovery under any other treatment. He was at the point of death when the nitrite of amyl was first administered, and no expectation was felt that he would rally under its use. It was ordered, because it was thought that he was entitled to the forlorn hope which it afforded, but almost with reluctance, because it seemed to be subjecting a remedy to an unfair trial, in making use of it under such extreme circumstances.

Isaac B—, aged 31, coal-miner, widower, was admitted from Wakefield, December 31st, 1864, having then become violent, in consequence of epileptic fits from which he had long suffered. He has all along been a most dangerous and intractable patient, especially at the time when his fits were due. Already debilitated by an attack of diarrhœa, he was assailed by the first of a series of fits on April 22nd, 1873. On April 23rd, he had three fits; on the 24th, four; on the 25th, three; on the 26th, five; on the 27th, ten; on the 28th nine; on the 29th, six; on the 30th, eight; on May 1st, eleven; and on the 2nd, sixteen. His condition then was simply desperate. All treatment had failed to moderate the severity of the paroxysms or to interrupt them. When visited at 11 P.M., he was pronounced to be dying. He had had five fits between 8 P.M. and that hour. He lay in a state of deep unconsciousness, and could not swallow. His head, face, and neck, were of a dull purple colour, and his skin elsewhere was dusky. Perspiration streamed copiously from his head and limbs. His breathing was loud and stertorous; his pulse 140, and his temperature 102·1° F. A piece of lint on which 10 minims of nitrite of amyl had been poured, was then held over his mouth and nostrils. In forty seconds he opened his eyes, in one minute raised his head, and looked about him. In two minutes he was tolerably conscious, and turned his head when called by name. In three minutes, 10 drops more of nitrite of amyl having been breathed, he swallowed a glassful of milk without difficulty. His pulse was then 150; his face redder and less purple than it had been before. In a little he relapsed into unconsciousness, but from that moment he had no more fits. An inhalation was given every hour during the next night and day, and recovery was unbroken. In ten hours he was quite conscious, and able to answer questions; and in a few days he was in his ordinary state of health, only perhaps a little more stupid and taciturn than he had been before the attack.

Thomas S—, admitted November 1st, 1866, age 40. Single, plasterer

from Bradford. Was reported as having been epileptic from childhood. On a former occasion, he had been an inmate of the West Riding Asylum for three years, and was brought back, because of noisy excitement with violence. During the six and a half years of this present residence in the Asylum, he has suffered from frequent fits, and from occasional attacks of excitement. He has gradually become more sullen and obstinate, and has latterly given expression to singular delusions, such as that his own head had been cut off, and another substituted for it. In the present year his fits have happened every three weeks with tolerable regularity ; four or five fits, of a severe type being spread over two or three days. Attacks begun on April 13th and May 4th, and on the morning of May 26th, a fit of great severity occurred at 8 A.M., and was rapidly followed by five other fits, one happening punctually every hour up till 1 A.M. After these fits, consciousness was not recovered, and the patient remained in a comatose state, breathing heavily, and incapable of being roused. Even reflex action was interfered with, touching the conjunctiva, and tickling the soles of the feet, producing no movements. At 1 P.M. he was made to inhale 6 drops of nitrite of amyl ; and seemed to awake up almost instantaneously. No sooner had a dusky flush spread over the countenance, than he opened his eyes and gazed vacantly about him. The inhalation was repeated every hour during the afternoon and evening, and after every inhalation a decided advance towards recovery took place. All coma and drowsiness disappeared, the patient grew more lively and talkative than is his wont ; partook of food, and sat up in bed. No fit occurred subsequent to the first inhalation. On the following day, May 27th, Thomas S— was in his usual state of health, but perhaps a little more intelligent than he has been for a year past.

The space at my disposal will not permit me to report three other cases, in which the *status epilepticus* was beneficially influenced by nitrite of amyl inhalation. At some future time I shall place these on record, and shall describe my further experience with this agent in the treatment of epilepsy.

ON THE LOCALISATION OF MOVEMENTS

CEREBRAL HEMISPHERES,

AS REVEALED BY CASES OF CONVULSION, CHOREA AND 'APHASIA.'

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EPILEPTIC AND PARALYSED.

THE results of the recent researches of Ferrier briefly stated by him, 'British Medical Journal,' April 26, 1873, and those of Fritsch and Hitzig, to which he refers, are highly important from several points of view. To me they are interesting, especially because they demonstrate in a novel and very striking way, the truth of what I have long urged, viz. : that discharges of *convolutions* develope *movements*, notwithstanding that destruction of limited parts of the brain produces no obvious loss of movements. The discharges I have studied have been those causing epileptiform seizures *in man*, and in particular those simple cases where the spasm developed begins unilaterally, in the hand or face or foot. The value of Ferrier's researches, and those of Fritsch and Hitzig, as showing by an *artificially* induced discharge of convolutions, the 'homologous convulsions' in lower animals is very great. The extreme importance of their facts for physiology proper, and for comparative anatomy, is too obvious for comment.

I have, however, written so many times on convulsions, and so much recently, that I cannot here, without undue recapitulation, consider the direct bearing of Ferrier's experiments on the anatomical and physiological part of our

Clinical Study of Convulsions. I may refer, however, to recent papers in the ' Lancet ' (January 18, 1873, *et seq.*), in which some of the points are illustrated. Epileptiform seizures are there looked upon as experiments on the brain made by disease, and as revealing to us, although necessarily *in the rough*, the localisation of special classes of movements in the cerebral hemisphere. I may refer also to a paper (' Brit. Med. Journal,' May 10) written since Ferrier published brief abstracts of his main conclusions, which is in great part devoted to a consideration of the direct bearing of his experiments on medical anatomy and physiology.

In this paper—partly preliminary to two others—I try to show (1) That convulsions, choreal movements, affections of speech, and other motor symptoms, are not *only* to be thought of as ' symptoms of disease,' but can be considered also as results of experiments made by disease revealing in the rough the functions of cerebral convolutions ; (2) To urge that the study of such *motor* symptoms is of direct importance for *mental* physiology ; (3) That if we do our work anatomically and physiologically, and reduce the very *different* symptoms to their lowest terms, to movements for example, we shall find that there are certain *fundamental* principles common to them all—common to symptoms so specially different as ' aphasia,' and a convulsion of one side of the body.[1]

The most important matter I have to urge, is the study of the localisation of movements on the *double* plan—by comparing the effects of destroying and discharging lesions on the brain of *man*. The following quotation, especially the parts I have italicised, will show my meaning on this point more plainly. It is the opening paragraph of a paper in the ' Lancet,' January 18, 1873.

' For some years I have studied cases of disease of the brain, not only for directly clinical, but for anatomical and physiological purposes. Cases of paralysis and convulsion may be looked upon as the results of *experiments made by disease* on particular parts of the nervous system of man.

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[1] Since writing the chief part of this paper I find that there is more recapitulation from former papers than I expected. I have acknowledged this in part by giving quotations from former papers and by foot-notes.

The study of palsies and convulsions from this point of view is the study of the effects of ' destroying lesions ' and of the effects of ' discharging lesions.' And for an exact knowledge of the particular movements most represented in particular centres, we must observe and *compare the effects of each kind of lesion.* It is just what the physiologist does in experimenting on animals ; to ascertain the exact distribution of a nerve, he destroys it, and also stimulates it. Indeed, *this double kind of study is essential* in the investigation of cases of nervous disease *for physiological purposes.* For limited *destroying lesions of some parts of the cerebral hemisphere produce no obvious symptoms ; while discharging lesions of those parts produce very striking symptoms.* By this *double* method we shall, I think, not only discover the particular parts of the nervous system where certain groups of movements are most represented (anatomical localisation), but, what is of equal importance, we shall also learn the order of action (physiological localisation) in which those movements are therein represented.' [1]

In the 'St. Andrew's Graduates'Transactions,' vol.iii. 1870, 'A Study of Convulsions,' I have considered convulsive seizures first from a clinical, and secondly from an anatomical and physiological point of view. I have indeed written on ' Convulsions beginning Unilaterally,' innumerable times during the last ten years, but I will only refer the reader to the two papers just mentioned. The earlier papers, and perhaps those above referred to as well, are full of crudities. It may be remembered in extenuation that my investigations of epilepsies have not been from orthodox points of view. I have not simply repeated accepted doctrines with slight variations and new illustrations. Working on a novel method, I run continual risk of making novel blunders. But in thinking for one's self there are certain kinds of blunders which almost must be made. And it is always easy to avoid appearing to go far wrong if one does not go far from the beaten track. Let me speak more particularly.

As will be seen stated at some length in what is to follow,

[1] On the Anatomical and Physiological Localisation of Movements in the Brain. 'Lancet,' January 18, 1873, *et seq.*

I do not in any case of convulsion, or occasional loss of con-
sciousness, endeavour to see if there be an approach to a
certain clinical standard—to ' genuine epilepsy.' I try in
each case to find the seat of the ' discharging lesion,' and also
the pathological process which has led to it.   The first
question in my mind is *not*, ' Is it a case of epilepsy ? ' but
' Where is the lesion permitting occasional excessive dis-
charge ? ' Hence the presentation of the facts to those who
do not look at cases of epilepsy from so novel a standpoint
seems strange and unreal.   My method is *just the opposite*
of the common method. It is a small matter to me whether a
case of convulsions or other paroxysmal nervous seizure is
to be called epileptic or not.   What I labour to find out is the
part of the brain of the functions of which the convulsion is the
brutal and sudden development.   For example, to quote from
a former paper,[1] ' We *do not care* to say that a tumour of the
brain (or minute changes near it) had " caused epilepsy," but
that changes in a particular region of the nervous system
—say in the region of the left middle cerebral artery—led to
convulsions, in which the spasm began in the right hand,
spread to the arm, attacked next the face, then the leg, &c.'

The new method has, I think, the advantage of showing
the relations of different ' diseases ' or symptoms.   This is
a matter of importance when symptoms are to be looked on
as the results of the experiments of disease.   To give
examples of the relations of different symptoms : there are
several *mobile* counterparts of hemiplegia. There is hemi-
chorea, there are certain cases of hemi-spasm, and there
is what I may call hemi-contracture, a mixture of palsy
and spasm. I call these one-sided mobile symptoms ' Hemi-
kineses.' I believe that each of them depends on disease
of the same *internal* region as does hemiplegia—the region
of the corpus striatum.   For the same *external* region is af-
fected in each.   To obtain a knowledge of the movements re-
presented in the cerebral district mentioned, we have to study
each one of these symptoms carefully. As some evidence
that this method of study is practically useful, I may adduce

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[1] ' St. Andrew's Reports,' vol. iii. 1870. After speaking of a case supposed to be
carefully investigated, on its own merits I remark as above.

Dr. Radcliffe's testimony. In his article on Chorea,[1] after stating the reasons I have advanced for the localisation of the changes producing chorea in convolutions near to the corpus striatum, he writes, ' for most assuredly the difficulties which beset any attempt to localise the choreic lesion in the nerve-centres, are not a little simplified by thus insisting upon the clinical relations between hemi-chorea and hemiplegia, as a ground for believing that the region of the corpus striatum is the part affected in both disorders.'

The old method, however, deals with circumscribed entities. It *looks* simpler. Thus many students I find are interested in being told that a case is one of *true* epilepsy, or of *real* chorea, or of *genuine* aphasia, who show no interest in the description of a paroxysm of convulsion, not even the simplest; who have never tried to form a clear conception of the *sort* of movements they can see in a choreal child, and who, whilst they are interested in such discussions, as whether a particular case is one of *genuine* aphasia or not, take no pains to obtain a realistic account of what that patient's condition was— what he really could say or could not say. They have been taught to study cases as the symptoms show *approaches* to certain clinical standards, and not as they show departures from healthy states. The latter plan (to illustrate by a simple case of convulsion) requires a more minute study of the paroxysms, and further, it demands, more *extensive* knowledge of *other* nervous diseases or symptoms. Those who only wish to know whether a given convulsion is epileptic or epileptiform, have an easy task. Those who wish to know whether the discharging lesion is of convolutions in the region of the middle cerebral artery or not, have a much more difficult task. They have to note carefully the muscles convulsed, in order to see if they are the same as those paralysed in cases of the common form of hemiplegia, which symptom is well known to be due to lesion in this region. And in order to be able to note this, they must know thoroughly beforehand, the symptoms of the several degrees of hemiplegia, as, for example,

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[1] Reynolds' 'System,' 2nd edit. vol. ii. p. 199. (Passage not in first edition.) I give comparison of hemiplegia and hemispasm p. 334.

that with a lesion of a certain degree of gravity there is deviation of the head and eyes.

One reason for the complexity of part of the work I have done in cases of epilepsy, chorea, &c., is that I have written of cases from several points of view. For example, not only have I urged that convulsions beginning unilaterally point to *disease* in the region of the middle cerebral artery— a clinical statement—but I have urged the study of them for purposes of Localisation of Movements—a physiological statement.[1] To make cases of convulsion of use for the latter purpose, much minute work and great patience are required. And after all the fear is that to many whose opinions are to be esteemed, it will seem a sheer waste of time to note the movements developed in a fit so minutely as is done, for example, in the following remarks [2] on a case of very partial convulsion. The extract, however, only gives a summary of what was observed in several seizures : ' The order of involvement was that the mouth all round was first in action, then the mouth was drawn to the left, then both eyes to the left (the head to the left), then the eyelids of both sides (the left the more) closed. The thorax was affected early, and the arm late.' [3]

But it is possible to observe many partial seizures pre-

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[1] See note 'Localisation,' 'Med. Times and Gazette,' August 15, 1868. See also St. Andrews' 'Reports,' vol. iii., 1870, from which I quote the following :—'In hemiplegia the loss is of a certain number of possible *simultaneous movements* of the face, arm, and leg—the sum of a number of possible co-ordinations in space. Similarly a convulsion on one side is the abrupt development of a certain number of possible *successions of movements* of the face, arm, and leg—the sum of a number of possible co-ordinations in time.'

[2] See ' Med. Times and Gazette,' January 6 and 27, 1872.

[3] It may be well to give the concluding paragraph of the remarks on this case, as they embody the main facts of the autopsy : ' In this case the lesion was not sufficiently local to enable one to conclude that fits beginning in the face show damage to any particular convolutional region. For instance, the fits may have been owing either to discharge of the grey matter of the convolutions of the temporo-sphenoidal lobe, or of the island of Reil. In most cases of convulsion beginning unilaterally the cerebral lesion is very extensive. I shall shortly, however, report a case of convulsion beginning in the left thumb, in which there was a tubercular tumour, the size of a hazel-nut, in the hinder part of *one* convolution—the third right frontal convolution. [See report of that case, ' Med. Times and Gazette,' November 30, 1872, p. 597.] By numerous observations of this kind we may confidently expect to arrive at clearer notions on localisation of movements.'

cisely, for some of them last a long time. I have witnessed one limited to the arm and face which lasted ten minutes.

Why should not a carefully observed convulsion of the right arm, associated with central disease so local as a tumour in the hinder part of the uppermost frontal convolution on the left side, be considered as an anatomical and physiological experiment, although a rough one, on part of the brain? But the idea of using cases of convulsions or of hemiplegia for anatomical and physiological experiments is entirely unfamiliar—and to many unfamiliarity is the same thing as unreality. I would here repeat what I have said in the ' Lancet,' January 18, to the effect that *there is no other way* of ascertaining the localisation of movements in the cerebral hemisphere *of man,* than by the study of his convulsive seizures. But let us take a simpler illustration. Let us take the very simplest of all cases of paralysis— hemiplegia from a *destroying lesion* of the ' motor tract.' Why should not the effects of an experiment which a clot makes on the corpus striatum *of man* be recorded in works on physiology, as well as the effects of intentional experimental injury to the corpus striatum of *a rabbit ?* It is our fault. The physiological part of our clinical work is not sufficiently methodical ; we have medical knowledge, and we have separately physiological knowledge, but our medical knowledge is not sufficiently physiological. On this matter I may quote from a lecture on hemiplegia published some years ago, and I do so none the less willingly because part of my own medical physiology in that lecture deserves criticism.

' — Our School Physiology and our Medical Physiology do not very well harmonise ; and this is particularly so of the corpus striatum and thalamus opticus. An eminent physician and physiologist, in his valuable text book of physiology, says that the corpora striata are not peculiarly concerned in movement, and he adds that " the recent experiments of Schiff, confirming and in many respects correcting those of Magendie and others, show that when they are removed in rabbits, sensation is unimpaired, *and the power of movement complete.*" But I suppose no physician denies that disease

of the corpus striatum in man produces paralysis of a great part of the muscles on the opposite side of the body.'[1]

To urge that the study of *convulsion* from discharge of *convolutions* is most important for *mental* physiology, as has been already done by implication, will seem more than strange to those who hold that the convolutions are parts of the 'organ of mind,' and are for 'ideas,' and that the subjacent 'motor tract' is the only part of the brain for movements.

This—the notion that the cerebral hemisphere being for ideas, consciousness, &c., is not for movements—accords with the prevailing theory of epilepsy. The motor symptom, the convulsion, is ascribed to discharge of the medulla oblongata, and the loss of consciousness—the mental symptom —to sudden and almost contemporaneous anæmia of another part, the 'organ of mind.' There are supposed to be necessarily *opposite* states of two separate parts—' une inertie totale (*cerveau*), et une suractivité fonctionnelle (*bulbe*).'[2] I shall give reasons for believing that all the symptoms in cases of 'epilepsy,' even as that term is used by the most rigid authorities, are owing to *one* state, one discharge, and that probably of a single cerebral hemisphere. (See pp. 328–9, and Appendix II., on Loss of Consciousness.)

In the case of partial convulsions instanced (p. 181), it is surely significant for mental physiology, that disease in the uppermost frontal convolution, did by some process, by any process, produce frequent occasional spasm of the arm; when it is remarked that the disease was seated deep in the 'organ of mind' (far from the motor tract), and that the part convulsed is the most specialised part of the whole body.

There seems to be an insuperable objection to the notion that the cerebral hemispheres are for movements. The opinion I have brought forward, that chorea may be due to disease of the corpus striatum, has received some favour (chiefly that of being disputed), but the suggestion that choreal movements may more probably depend on disease of *convolutions* near to that centre has scarcely been mentioned. The reason, I suppose, is that the convolutions are con-

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[1] 'Lond. Hosp. Reports,' 1865, vol. ii. p. 301. See Appendix II. to next paper.

[2] 'Jaccoud, 'Pathologie Interne,' p. 386.

sidered to be *not* for *movements* but for *ideas.* In another paper I hope to show clearly that there is no contradiction in supposing that the convolutions are for ideas and for movements too. I shall try, indeed, to show that *sudden discharges* give *proof* that sensori-*motor* processes are the *anatomical substrata of ideas.* And in the case of chorea, if the very great speciality both of the parts most moved and of the movements themselves be considered, there is nothing unreasonable in supposing that the sensori-*motor* processes which are affected are of *convolutions* in the region of the corpus striatum and are those for 'ideas' of touch, weight, &c., in *acquiring which ideas,* movements of the hand and arm were necessarily concerned. It is, indeed, most important to note the great speciality of the movements in cases of chorea; they should not be dismissed as 'disorders of co-ordination,' as if there were a 'faculty' of co-ordination. Wherever nerve-fibres meet in ganglion cells, there is a centre of co-ordination; what is anatomically a sensori-motor process, is physiologically 'a co-ordination.'

The great speciality and the separateness of the movements in cases of hemi-chorea is quite as strong evidence of their dependence on discharging lesions of very highly 'evolved' centres (convolutions of the brain), as is that supplied by the fact that the parts affected (face, arm, and leg) are the parts affected in hemiplegia, which symptom is undoubtedly owing to a destroying lesion of a very highly evolved centre. The following quotation [1] shows that these opinions are not hastily formed :—

'It is not denied that "disorderly movements" occur with disease in many parts, probably in most parts, of the nervous system. What I wish to show is, that *certain* irregular movements—often affecting the face, arm, and leg of one side only—occur from disease of the higher centres of movement, viz., *of the convolutions* near to *the corpus striatum.* It is especially to be observed that they differ from the jerky movements of the arm occasionally seen in severe cases of locomotor ataxy. They are not mere spasms and cramps.

[1] 'Edin. Med. Journal,' October, 1868.

They are an aimless profusion of movements of considerable complexity, much nearer the purposive movements of health.'

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' Now it is clear that close upon the corpus striatum lie the rudimentary arrangements of fibres and cells for the highly complex and widely associated movements of speech, and it is, I think—independently of other arguments—at least plausible that corresponding movements of the arm—*which may be called, according to our stand-point of thought, either rudimentary psychical, or highly developed physical movements —should have their centres here too.*'

' Although I have instanced series of comparatively simple movements of single parts analogous to ataxy of articulation, there are, in some cases of chorea, *movements* of much wider range, implying, I imagine, changes deeper and wider [?] in the hemisphere, just as there are probably movements (i.e. misuse of words or incoherence) dependent on changes deeper in the left hemisphere than the parts close to the corpus striatum.'

.   .   .   .   .

' But we must begin our studies of *mind* by a consideration of the more rudimentary phenomena, *although we should not make arbitrary distinctions betwixt those which are grossly motor and those sensori-motor impulses which we speak of as being mental.*'

It will probably seem to some a very abrupt change to speak of ' aphasia ' after speaking of convulsion and chorea. I think myself it is the very relation in which the fundamental nature of loss of speech can be most methodically considered.   In some cases of convulsion beginning unilaterally there is affection of speech.   But this is not the best reason for considering them together.   The reason is that in affection of speech there is an affection of a *highly special class of movements*, exactly as there is of other special classes in cases of convulsion and chorea.   Observations of cases show that with *right* hemiplegia there *are* all kinds of defects, from ataxy of articulation to mistakes in words. Both are really motor defects.   There is no *abrupt* dis-

tinction made by disease betwixt articulatory movements and words, and it is not well to make a clinical entity of aphasia by paring away a difficulty of articulation at one end of a series of defects and incoherence at the other end.

The following extract [1] will show that changes of opinion on this matter were forced on me by observations of cases of disease. I give the quotation as I wish to point out that the opinions I here put forward have not been formed hastily :—

'When my attention was first drawn to the class of cases (Class II.) I am discussing, I thought there was a fundamental distinction betwixt the mistakes of words and " ataxy " of articulation—the mistakes of muscular movements. I used to suppose there was a part for words, and, besides, a distinct co-ordinating apparatus for the movements of words, and that Broca's convolution was a sort of cerebellum for articulation. It is quite true that the defects I have recently mentioned ("ataxy " of articulation and mistakes in words) are very different, but then they are not altogether different. It was forced on me that the separation was one which observation of cases did not justify, and I soon concluded that, to use a crude expression towards the end of my first paper,[2] " the ataxy of articulation was a quasimental defect—an inability to combine muscular movements in a particular mental *act*." Again, in a subsequent paper,[3] speaking of defects accompanying right hemiplegia, I remarked that " it was hard to say where obviously motor symptoms ended and mental ones began." I have long believed it to be not only hard, but impossible, even using the words " motor " and " mental " in the popular sense. I now think that the only differences in ataxy of articulation, mistakes of words, and disorder of ideas are differences of " compound degree." '

It would be marvellous if there were *not* all degrees of defects. For is it not accepted doctrine that nervous processes *gradually* increase in complexity, in independence,

[1] 'Med. Times and Gazette,' September 26, 1868.

[2] 'Lond. Hosp. Reports,' 1864, p. 471.

[3] 'Med. Times and Gazette,' January 28, 1865.

and in specialty? If so, is there anything at all wonderful in the fact that damage low down in the series of sensori-motor processes (close to and of the corpus striatum) should produce a defect of a comparatively simple nature—ataxy of articulation—and that damage high up in the very same series should produce one of a highly elaborate nature—' loss of memory ' for words?

In friendly controversies my opponents imply (because I say that from a physiological point of view a certain defect of *articulation* is a *rudimentary* defect of *speech*, or that ' loss of memory for words ' is loss of sensori-*motor* processes for words) that I am not aware of the differences betwixt the two defects.[1] Attempts are made to assist my imagination by such remarks as, ' *That* contains an *idea* ; the other is the agent, executive, &c.' I do not really think I particularly need *these* helps. I used to make that distinction myself, as the quotation given (p. 185) shows. A little consideration would make it plain that it is impossible for anyone to overlook the difference betwixt a sort of stammering talk and saying ' orange ' for ' onion.' But is it not just possible to trace *fundamental* resemblances without ignoring exceedingly striking and very superficial resemblances. Do I not know as well as an ignorant man that a whale is *very* unlike a bat, although I steadily insist that in fundamental characters they are far more alike than a whale and a fish ? And is it not possible without contradiction to hold that an ataxic articulation and a mistake in a word are *fundamentally* defects of the same kind and yet *very* different in specialty ? Is it not likely *à priori* that the two symptoms are owing to damages of *one* series of sensori-motor processes—the damage being of sensori-motor processes respectively low and high in evolution.

Psychology is older than Mental Physiology, and thus

[1] It ie very important to bear in mind that, besides paralytic difficulty of arti-culation from disease of the nerve trunks to the articulatory muscles, or of their immediate centres in the medulla-oblongata, there is a difficulty of articulation (I call it ataxic) in which paralysis of the lips, tongue, and palate is not discoverable. In the latter the patient has usually some difficulty in expressing himself in writing —showing that the ataxy ie a rudimentary defect of *speech*. It occurs with right hemi-plegia. I have eeen it in one case of disease of the pons Varolii.

when we come to that arbitrarily limited part of medical physiology which deals with defects of mental operations, we use psychological terms, not only to describe but also to explain abnormal physiological phenomena. But in order to make our work at cases of aphasia a *methodical part* of our work at other cases of disease of the brain, we must reduce the symptoms to their lowest terms—to the same terms to which many of the coarser symptoms are reducible, that is to say, to movements (sensori-motor). We must study affections of speech, so far as is practicable, in the same manner as we do cases of chorea, convulsion, and hemiplegia. The distinction into ' loss of memory for words ' and ' loss of movement for words ' (speaking of cases of damage to the brain), looks clear and orderly, but it will not work. On the other hand, to say that there are all degrees, from difficulty of articulation to mistakes in words (and there are), looks indefinite. But there need be no trace of indefiniteness in the work to be done except what is the outcome of the workings of our own minds. The defects of speech occur in *cases* which can be investigated with any degree of minuteness.

As some evidence of the merging of the several defects into one another, one listens with interest to discussions on cases of defects of speech. One speaker will take the view —the patient being present—that the defect is *only* one of articulation, another urges that there is at least *some* aphasia. Is not this good evidence that at the lower extreme the defects merge ? Again, it is occasionally discussed whether a man's abnormal way of talking is of an aphasic character or is owing to a mental defect. Does not this show that at the other extreme there is no abrupt demarcation ? Does it not show that a person has not mind *and* speech, any more than he has speech *and* movements of articulation ? There are no absolute separations betwixt either. All phenomena of the organism—whether they be such as walking or such as thinking—have for their anatomical substrata sensori-*motor* processes, and for the condition of their development excitation of nerve-cells and fibres.

Disease *appears* occasionally to respect the two divisions ; there are cases of little more than ataxy of articulation, and

cases of mistake of words only. They appear to be very distinct things. The explanation, however, is, I believe, simply that different pathological processes tend to damage different parts of the region of the corpus striatum. Thus either clot or softening (from embolism or thrombosis) may produce entire loss of speech. But what is to the point is that each of them can, when the damage is limited, produce *defect* of speech. Now if there be *defect* of speech with softening, it, as I have long noticed,[1] more often consists of mistakes in words. But in cases of cerebral hæmorrhage, the defect of speech (if it is only defective) is more likely to be one of articulation (it is an ataxy of articulation). In the former case the pathological process is one which can affect convolutions directly, and that without at the same time extensively damaging the motor-tract; the pathological process in the latter affects chiefly the motor-tract (as we are considering *defects* of speech we are concerned with *limited* damage). But to speak of convolutions and motor-tract here is not to speak in terms sufficiently safe. It encourages the notion I am trying to disprove throughout this article, that there is an abruptly separate part for *ideas* and another separate part for *movements*. (The anatomical substrata of ideas can be nothing other than processes for impressions and movements.) We now say, then, that whilst in each kind of defect the very same series of sensori-motor processes is damaged, there is in the former (mistakes in words) damage where the series is comparatively high in evolutions, and in the latter (ataxy of articulation), where it is low in evolution. To the above differences correspond important clinical differences. In the former, if there be hemiplegia, it is usually slight and transitory; in the latter the hemiplegia is usually decided and persistent. The bearing of these two facts is obviously in accord with what has been said. Another fact, I think, is, that the more the leg and the less the arm suffer in cases of right hemiplegia, the less defect of speech there is.[2]

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[1] 'Med. Times and Gazette,' June 23, 1866, p, 662.

[2] I have put forward the opinion that mistakes in words are analogous to choreal

The objection that when we speak internally, as, for instance, when we express ourselves in writing (and we must speak before we express ourselves in writing), there *is no movement* of the articulatory organs, is not of weight. To remember a word is to have a faint excitation of the sensori-motor process of that word.[1] The objection tacitly assumes that the convolutions, being for *ideas*, cannot be for *movements*. It is only supposed that in internal speech there is *faint* excitation of those *central* sensori-motor processes which for external speech require to be strongly excited. There is as much speech when I say ' Gold is yellow ' to myself as there is when I say it aloud. We cannot surely suppose that different sets of sensori-motor processes are concerned when we ' think ' ' Gold is yellow ' and when we ' talk it.' Of course there is a vast difference in the degree, and, what is equally important, in the extent of the excitation. In one there is a ' motor impulse ; ' in the other, actual movement. Perhaps the most striking way of putting it will be to assert that the patient who has lost power over his articulatory organs from disease in the medulla oblongata, and who cannot, therefore, get a word *out*, has no affection of speech. The proof is that he continues able to express himself in writing, *and he could not write unless he spoke internally.* There is, indeed, no more reason why a patient who has become unable to *talk* because his tongue, lips, and palate will not move, should not *speak* internally than there is that a person who has *become* blind should not see *internally* (' ideally,' ' subjectively,' &c.). And that the man who has be-

movements in so far as this ⎯I mean here that they are the results of *over* action of nervous processes, and thus that they (degrees of *disorders* of speech) differ fundamentally from degrees of *loss* of speech, the analogues of degrees of paralysis. This opinion I do not discard. But I now think, as was several years ago suggested to me by Mr. Herbert Spencer, that there is in some of the cases of mistakes in words, the mistake of making a more general for a more special symbol. Thus, a patient seeming afraid of a kitten put on her lap, said, ' Take little fur child away.' When so, the cases evidently come in the category of palsies ; they conform to the law of destroying lesions—the patient is, in his speech, ' reduced to a more automatic condition.' The analogues of choreal movements will be the utterances of more special for more general symbols.

[1] See Principles of Psychology, first edit., p. 359 ; Spencer on Memory, &c.

come blind does see *internally* by central excitation is plain, since he dreams of objects. Doubtless (although I have put no questions on the matter to such persons) those who have lost articulation, but not speech, dream of talking (aloud).

The following quotations concerning Laura Bridgeman bear on several foregoing matters. The first of them I have used before [1] to show that there is good authority for the belief that in deaf-mutes a set of educated *movements* (those of their hands) serve as our articulatory movements do in internal as well as in external speech. It was used also to show that, in deaf-mutes, there are very special ('mental') movements of the hands, which serve immediately in mental operations. I do not, however, suppose it to be likely that deaf-mutes think in images of 'raised letters,' if that be what the archbishop means. This and the quotation from Whately also show that *central* excitation of nervous processes for hand movements suffices for the internal speech of deaf-mutes—motor impulses instead of actual movements.

Laura Bridgeman, when she dreamed, ' talked ' to herself in finger-language, and doubtless she thought by aid of the nervous processes for signs she made with her fingers. As we generally think by help of symbolic movements of our articulatory organs, or rather of the nervous impulses to move them, so she thought by help of symbolic finger-language.

Dr. William Thomson, the present Archbishop of York, writes: [2] 'Those among the deaf and dumb who have been taught by the pains of an enlightened humanity to converse and to think, must use, instead of the remembered words which we employ, the remembered images of hands in the various combinations of finger-speech [here the italics are mine] *as the symbols of their thoughts*. The deaf and blind, taught the names of objects from raised letters, must think, not by associations of sound, but by touch.'

Whately, speaking of Laura Bridgeman, says :—

' The remarkable circumstance in reference to the present subject is, that, when she is alone, her *fingers are generally observed to be moving*, though the signs are so slight and im-

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[1] ' Med. Times and Gazette,' June 23, 1866, p. 662.

[2] ' An Outline of the Necessary Laws of Thought.'

perfect that others cannot make out what she is thinking of. But if they enquire of her, she will tell them.

'It seems that, having once learnt the use of *signs*, she finds the necessity of them as an *instrument of thought*, when thinking of anything beyond mere individual objects of sense.

'And, doubtless, everyone else does the same; though in *our* case no one can (as in the case of Laura Bridgeman) *see* the operation; nor, in general, can it be *heard*; though some few persons have a habit of occasionally audibly talking to themselves, or, as it is called, "thinking aloud."'

There is, from another department of clinical medicine, proof, although indirect, of the opinion that faint and central excitation of sensori-*motor* processes suffices in thought. It is necessary to give this at some length, as we shall thus obtain facts bearing on the way in which *movements* serve in higher mental operations, facts which will make clearer what has been said on the relation of 'mental' to 'physical' operations.

In a case of sudden paralysis of the right external rectus, when the patient tries to turn the eye outwards, that is, to the right, 'l'organe n'obéit plus à la volonté, il s'arrête à moitié chemin et les objets *paraissent se déplacer à droite, bien que l'œil et les images rétiniennes qui s'y produisent ne changent pas de position*.'[1]

In a case of *paresis* of the external rectus there is evidence of desorientation; in striking at an object held on the paralysed side, the patient's hand goes beyond the object. To bring the eye upon the object, the patient has to 'send down' as much 'force' as would in health be enough to carry the eye beyond it. In the case supposed, the defect is in the conducting nerve trunk, not in the energising centre. It is in the transmission of force, not in there being a less amount for transmission; an unusual amount *is* transmitted part of the way, but it cannot all get to the muscle. The patient

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[1] 'Helmholtz.' French translation, by Javal and Klein, of the 'Physiological Optics,' p. 764. (No italics in original.)

judges by the strength of the central excitation, not by what that actually accomplishes.[1]

Of the case of *paralysis* Helmholtz says : 'L'acte volontaire *ne se traduit par rien en dehors du système nerveux*, et cependant nous jugeons la direction de la ligne visuelle *comme si la volonté avait exercé ses effets normaux*,' op. cit. p. 764. (No italics in original.)   These facts seem to me to give striking corroboration of the view that central[2] excitation suffices for internal speech, and indirectly that there are in internal speech, or ' memory of words,' motor impulses— rudimentary or incipient movements.

We must now remark very briefly on certain other ocular motor symptoms, with the same intentions.   (I assume that the reader is well acquainted with all the symptoms I speak of.   They are nearly all common ones.)

In some cases of hemiplegia there occurs a symptom which shows that there are in the brain, at any rate in the corpus striatum and adjoining convolutions, sensori-motor processes for highly special movements of the two eyes.   These very special ocular symptoms are conveniently considered after speaking of the strange ' mental ' effects occurring in cases of palsy of a nerve *trunk*.   The symptoms are, turning of the two eyes *from* the side paralysed in hemi*plegia*, and, correspondingly, turning of the two eyes *to* the side convulsed in cases of hemi-*spasm*.   This lateral deviation of the eyes (first described by Vulpian and Prévost[3]) seems to me to be the motor analogue of hemi-opia.   The ocular movements lost are *as special* as the articulatory movements lost in certain cases of affection of speech.   Put otherwise we say that lateral devi-

[1] These facts explain the patient's giddiness, in the production of which, contrary to what is commonly supposed, double vision has little share.   Vertigo, I would define to be taking as accomplished a movement which is only strongly attempted—in which there is only a strong or wide *central* excitation.   This is the physiological definition; the pathology of vertigo is another matter.

[2] If we impress the retina with a scarlet object, and thus obtain a negative image, that negative image will appear to us to enlarge as we look into the distance.   Under atropine, objects appear to be smaller.   One of my epileptic patients told me to-day that when his fit sets in, the wall seems 'to come nearer,' and, again, that distant objects seem ' clearer.'

[3] In this country by Humphry, Lockhart, Clark, Hutchinson, Broadbent, Russell Reynolds, and myself.

.ation of the eyes differs as much from paralysis of the third or sixth nerve, as ataxy of articulation does from defective articulation owing to paralysis of the tongue, palate, and lips.

The occurrence of this symptom from disease of the brain is of very great importance for mental physiology;[1] *movements*[2] *of the eyes enter into the anatomical substrata of our visual 'ideas.'* The significance of the occurrence of this symptom (in severe lesions and strong discharges) along with affection of our chief tactual organs, is very great. Donders supposes, with Hering, that there are movements of the eyes together for *direction* (upwards, downwards, inwards, and outwards), and also of adduction and abduction for *distance*. He shows from Adamük's[3] experiments that ' au moins chez le chien et chez le chat, les deux yeux ont une innervation commune, qui part des tubercules antérieurs des corps quadrijumeaux. L'éminence droite régit les mouvements des deux yeux vers le côté gauche, et vice versâ. En irritant des points différents de chaque éminence, on peut provoquer le mouvement dans une direction quelconque, mais toujours les deux yeux se meuvent simultanément et en conservant entre eux une relation déterminée.' But such movements are also represented in the corpus striatum and adjacent convolutions (perhaps I should say, re-represented), *and in direct relation with movements of our chief tactual organs*. The movement is lost in hemiplegia, and is developed at a certain stage in convulsion beginning unilaterally. I was long puzzled by the fact that in the lateral deviation of the eyes in hemiplegia and convulsions beginning unilaterally the eyes were parallel. As the act of accommodation (in which the eyeballs are con-

[1] I am surprised that Vulpian's important statements on this ocular symptom have received so little attention in this country. Lateral deviation of the two eyes is valuable clinically as evidence of a gross lesion in cases of apoplexy; enabling us sometimes to tell cerebral hæmorrhage from drunkenness (Prévost). I have found it a most important help towards completing the parallel betwixt hemiplegia and hemi-spasm (see 'Lancet,' February 16, 1867). Thirdly, as suggeste in the text, the symptom is one of extreme importance for mental physiology.

[2] See Spencer's 'Psychology,' second edit., vol. i. chap. xiii.

[3] I take these extracts from a translation of a paper by Donders in ' Robin's Journal,' September and October, 1872. In a very brief statement in the 'Lancet' (February 15), of what is given at more length above, I represented Donders as speaking only of the *lateral* movements of the eyes.

verged) is a very important one, I expected to find the
movement of convergence the *first* of the ocular movements
to suffer.   For, as I shall mention more particularly in my
next article, the most special movements suffer first in cases
of cerebral lesions (pp. 314–15).   But I now see that conver-
gence, as it has to do with distances, belongs to the loco-
motor series (cerebellum) and not to the tactual series
(cerebrum).   The movements for carrying the retinæ over
objects, are just as special in the cerebral series as those for
convergence are in the cerebellar series.   I say ' cerebellar
series,' because my inference has been that the movements
of convergence are chiefly represented along with movements
of our spine, arms, and legs for locomotion.   At any rate,
according to Donders and Adamük, the two kinds of move-
ments are *differently* represented in different parts of the
corpora quadrigemina.   I continue the quotation, ' Par l'irri-
tation de la partie postérieure, soit de l'éminence droite, soit
de l'éminence gauche, on obtient une forte convergence, avec
abaissement simultané des lignes visuelles et rétrécissement
de la pupille.'   In the corpora quadrigemina we should not
expect any very great differentiation of the two classes of
movements, any more than of movements for articulation
and deglutition in the medulla oblongata.   But betwixt the
mode of representation of movements in the cerebrum and
in the cerebellum, we should expect the differentiation to
be carried to its extreme.[1]

In healthy looking at near objects, of course, both classes
of ocular movements will be developed.   The eyes must be
adjusted; when ' fixed together ' so as to ' reach ' the object,
they can be carried over the object; the retinæ can then
' feel ' it.

I have urged the doctrines stated in this paper many
times ; I have urged them in places where at first glance they

[1] As will be pointed out later from larger illustrations, it does not follow that
movements represented in a lower centre, are not also represented in the higher
centre.   Are we to believe that the movements by which a headless frog rubs
vinegar off its back, are not also represented, and that more specially in the
detached head ?   Here the fact I have insisted on, that discharging lesions
produce motor symptoms when destroying lesions of the same parts do not,
obviously bears closely.

seem out of place, as in the Ophthalmic Hospital Reports.[1] But the object I have had has been one. I have wished to show that there are for those who study muscular disorders of the eye, for those who study hemiplegia and chorea, and for those who study diseases of the mind certain simple and yet fundamental principles, which give a harmony to their very different facts. If we work anatomically and physiologically (not at clinical entities) we shall, I think, make more progress than we do; we shall work together without confusion. Above all, to make our studies of mental diseases part of our scheme of studying nervous diseases in general, our method must be anatomical and physiological— not psychological. I conclude this paper by a quotation from an article I wrote some years ago. ' Special work at diseases of the mind should, I feel convinced, be begun only after a large real experience of all the special phenomena of motion and sensation that damage to any parts of the nervous system may give rise to. I say a real experience, as I suppose a collection of numberless facts, however accurately gathered, is not held to be of itself a real experience. Unless a man can put the particular phenomena he himself sees under more general laws, or unless he tries to do this, he can scarcely be said to know or to be studying a thing in any very valuable sense. The knowledge the ophthalmologist has of muscular disorders of the eye, the knowledge the physician has of defects of articulation, of chorea, and of epilepsy, and that the psychologist [alienist physician I should have said] has of incoherence and delusions, should aim to be Physiological Units,[2] each different, but each related to a wide common knowledge of such laws—present or in progress— as those of the Evolution of Sensation and Movement in organisms.' [3]

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[1] *Vide infra.*

[2] Physiological unit is a term introduced by Mr. Herbert Spencer. (See his ' Principles of Biology.')

[3] ' Royal Lond. Ophth. Hosp. Rep.,' vol. v. part iv. 1866.

ON ELECTRO-EXCITABILITY

IN

MENTAL AND NERVOUS DISEASES.

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Of late years electricity has been largely used in our hospitals and asylums, and its value in the hands of the physiologist and of the physician is every day becoming more and more apparent. A good deal has been written concerning the therapeutic uses of its various forms, and numerous researches have been made as to its physiological actions since the publication of the elaborate investigations of Duchenne.

I am not aware, however, that any extensive observations have been made as to its diagnostic value in the various disorders of the nervous system to be met with amongst the inmates of a large hospital for the insane.

The experiments described in the following Paper were undertaken in the hope that the results obtained would be of service in assisting to elucidate the diagnosis between various forms of mental and nervous disease in their earlier stages, and in cases of an obscure character.

The experiments — which were of a very tedious and laborious nature—were very carefully carried out, at the cost of much time and patience; and the chiefly negative character of the results is, therefore, perhaps somewhat dis-

heartening. Nevertheless, I do not hesitate to publish them; and still believe that, with machines provided with the means of more accurately determining and regulating the force and tension of the current than those I had the opportunity of using possessed, facts of greater importance may yet be elicited.

It is well known that many of the aids we possess as practitioners of the healing art have suffered from being too much lauded when first introduced. Less than half a century ago there was a strong and wide-spread prejudice against the use of the stethoscope as an aid to the diagnosis of diseases of the chest, arising from the fact that those who introduced it into this country undertook to arrive at a correct diagnosis by its use alone, without taking into account the symptoms of the maladies with which they had to deal. Consequently, they were frequently in error, and the result was, that they brought discredit upon the use of an instrument which has proved to be invaluable in the hands of the skilled physician, and defeated the end which they sought to attain. The same is true of electricity. More than fifty years ago the results of its use as a curative agent were reported to be most marvellous—a few individuals, too zealous in its cause, holding it up to the profession as the remedy for all diseases. It was put to the test, but, failing to come up to the expectations of those who gave it a trial, was cast aside for a time as being worthless, or of little value.

Before long, however, fresh enquiries and more careful experiments and observations began to be made regarding it. In 1839, Dr. Marshall Hall, in a paper published in the ' Medico-Chirurgical Transactions ' for that year, drew attention to its diagnostic value in the different forms of paralysis ; and, in 1841, a paper by Dr. Golding Bird appeared in ' Guy's Hospital Reports,' giving the results of its employment in Chorea, Paralysis, and Amenorrhœa. In the same year Dr. Pereira made some observations upon the effect of the electric current in cases of Paralysis; and, in 1845, Dr. Copland made a similar series of experiments. Dr. Todd, also, made some investigations into the contractility of muscles in cases of paralysis, criticising Dr. Marshall Hall's

paper; and, in 1850, M. Duchenne, of Boulogne, published a paper in the 'Archives Générales de Médécine' on the same subject. It was Duchenne's famous work, however, on localised electrization which did most to renew the employment of electricity in medicine. To it we owe almost all that is known regarding the local effects produced by static and dynamic electricity, and by the induced current; more especially regarding the action of the latter upon the skin, the muscles, and the nerves.

Duchenne has proved that it is possible to limit the electric current to different tissues and organs of the body; and he has also demonstrated that contraction can be produced in a muscle by direct application of the current to its substance, without the intervention of its motor nerve. For producing this electro-muscular contraction, the faradic current is, of the three forms of electricity, the most suitable. ' Of the three kinds of electricity, induced electricity is the most fitted for muscular electrization, especially when that operation requires to be practised frequently, and for a long time . . . Thus faradisation excites the most powerful muscular contractions, without at the same time powerfully exciting the cutaneous sensibility, without producing shock, without throwing the organs into a kind of stupor, without rupturing capillary vessels; in a word, without occasioning, like static electricity, any accidents from which the patients might recover with difficulty. . . . We have, therefore, good reason to say that faradic electricity, which in no way alters the tissues, is essentially medical electricity.' [1]

Of the two kinds of faradic electricity—electro-magnetism and magneto-electricity—the former is much more suitable for medical purposes than the latter, the old drawbacks to its employment having in great measure disappeared with the improvements in the machines which are now in use. The effects produced by it are also much more easily borne than those produced by magneto-electricity.

[1] ' A Treatise on Localised Electrization, and its Applications to Pathology and Therapeutics,' by Dr. G. B. Duchenne. Translated from the third edition of the original by Herbert Tibbits, M.D.

Of the induction machines to be had at a moderate cost, those invented by Stöhrer, of Dresden, are about as good as any at present in use for medical purposes. As all my experiments were made with these instruments, a short description of their construction and mode of action will not be out of place here, as a knowledge of some points concerning these is necessary in order to follow me in my further observations. I find both the larger and the smaller apparatus so well described by Dr. Herbert Tibbits in his work on medical electricity recently published that I cannot do better than quote his description *in extenso.*

'The battery of this instrument consists of carbon and zinc, without an earthenware cell. The carbon, hollow within, filled with sand, and closed by a glass stopper, serves for the reception of a concentrated solution of chromic acid in water. Of this solution 10 or 12 drops should be added whenever the battery is recharged. The zinc surrounds the carbon, but is kept from contact by glass insulating buttons. These elements are placed in a circular glass cell, which serves for the reception of diluted sulphuric acid (one part acid to seven parts of water). This cell is so arranged in the completed apparatus, that it can be moved vertically up and down, and can be fixed at any point. By this arrangement the acid can be brought into contact with the whole, or with part of the zinc and carbon, or by shutting down the glass, can be excluded from them altogether. In the latter case, the acid will only fill the lower third of the glass. The advantages arising from being able to remove all the elements at once, and without difficulty, from the exciting fluid, and from the facility with which they may be brought into action, are obvious. Stöhrer constructs a smaller and a larger instrument. The battery of the former is constituted by a single cell; of the latter, by two cells, which may, however, be arranged either as two pairs, or as a single pair of elements. Both possess a primary and a secondary coil, the currents of each of which can be made use of separately; and in both the currents have a definite direction, positive electricity being set free at one terminal, and negative at the other, of each of the

coils. The terminal from which the positive current pro-
ceeds may be ascertained by the decomposition of iodide of
potassium. The larger instrument differs from the smaller
in having a much greater range of power, more thorough
means of graduating the currents, and a more elaborate
arrangement of the interrupting hammer. With practice,
however, the force and rate of interruption of the smaller
instrument may be regulated with much nicety. To neither
instrument is a water graduator attached ; but if needed for
any special nicety of application, one can readily be added,
and it would be best carried loose in the drawer for acces-
sories, to be attached only when required. Graduation of
the strength of the currents is effected by the arrangement
of the coils. The primary coil is fixed upon a pedestal ; the
secondary is movable, and is brought into and placed out of
action by being lifted over or thrust away from the primary.
The degree of action in the secondary coil is proportionate
to the extent to which it is brought under the influence of
the primary. The action of the primary coil is regulated in
the smaller instrument by the extent to which it is masked
by the secondary coil—the latter acting upon it as a metallic
sheath would do. In the larger apparatus a special copper
sheath is provided for the graduation of the current of the
primary coil. It is to be regretted that a similar arrange-
ment has not been adopted in the smaller instrument.' [1]

To this description I have only to add, that in both
instruments, the secondary coil has attached to it a rod
bearing upon it a series of transverse lines, which are
numbered from below upwards—the numbers commencing
when the secondary is fully under the influence of the
primary coil ; so that a higher number indicates a dimi-
nution in the strength of the current. The frequency of the
interruptions of the current is regulated by means of a screw,
and, by practice, can be determined with considerable nicety
by means of the musical sounds produced by the interrupting
hammer.

As Duchenne has pointed out, the current of the primary

[1] 'A Handbook of Medical Electricity,' by Herbert Tibbits, M.D., L.R.C.P.,
London, 1873, pp. 38–41.

coil of an induction apparatus has less effect upon the sensibility of the skin than that of the secondary, but excites to a greater extent the sensibility of the muscles, as well as their contractility. When moistened electrodes are employed, the current acts upon the muscles and nerves without having any great effect upon the skin, it being only at the moment of contact of the electrodes with the skin that any cutaneous sensation produced by the current is felt. When it is desired to operate upon the muscles, the secondary current, therefore, is much more easily borne than the primary, after the electrodes are fairly applied. For this reason, as well as on account of the graduator attached to it, I have used almost exclusively the current of the secondary coil in making these experiments, the current of the primary coil having been used only in a few exceptional instances. A current of rapid intermission has also been employed throughout. In order to faradise singly each of the facial muscles, I constructed electrodes specially adapted for this purpose. They each consist simply of a piece of medium-sized copper wire about a foot in length, bent to a suitable curve, provided with an insulating handle, and covered at the point with wash leather. I have found these to answer very well for operating upon the facial muscles. For most of the other muscles the electrodes belonging to the instrument have been used. In the practice of localised muscular faradization it is necessary to attend to the following amongst other precautions :—

(1). Care must be taken to avoid the nerve of supply of the muscle being operated upon.

(2). In comparing the electro-muscular excitability in different cases, the electrodes should be placed as nearly as possible over the same points of the same muscle in different individuals.

(3). In operating upon the facial muscles, care must be taken to avoid placing the electrodes over the infraorbital or mental nerves, as acute pain is the result if these nerves are acted upon.

(4). It is important that the wash leather covering the ends of the electrodes should be thoroughly saturated before

beginning to faradise the muscles, when a comparison is being made of their electro-contractility. Otherwise the results will not be trustworthy, as it will require a stronger current to produce contraction in the muscles first operated upon.

I now proceed to give the results of the experiments I have made with regard to the electro-excitability in various mental and nervous diseases. Part of these were carried out in this asylum, and the remainder in the West Riding Asylum, Wakefield. For the former, the instrument used was Stöhrer's smaller induction apparatus, and for the latter, the larger apparatus by the same inventor. I will first give the results of the examination of the muscles of four healthy men (upon whom I operated in order to get a standard for comparison), and then give the results obtained in cases of general paralysis of the insane, epilepsy, mania, dementia, chronic disorganisation of the brain, progressive muscular atrophy with insanity, melancholia, hemiplegia, and locomotor ataxy.

In all the tables the numbers are in the inverse ratio of the power required to produce contraction; and each number represents the *lowest* power of the current of the secondary coil required to produce contraction in the muscle. When no result is recorded, the application of the electrodes to the muscle in question was found to be impracticable, owing to the restlessness of the patient, or to some other cause. The letter 'n' signifies that no contraction could be produced by means of the highest power of the secondary current of the instrument used. It must be understood that the numbers on the larger instrument by no means indicate the same force of current as the corresponding numbers on the smaller. All the tables refer to the electro-excitability of the *contractility* of muscles, and when the term *excitability* alone is used, this is understood. The term *irritability* is used synonymously.

Electro-excitability of the contractility of the muscles in :—

1. *Sane men in ordinary health.*—

TABLE I.

Instrument used = smaller apparatus.

Name of muscle	W. F. 1st sitting	Do. 2nd sitting	Do. 3rd sitting	Do. 4th sitting	M. B. 1st sitting	Do. 2nd sitting	Do. 3rd sitting	T. L.
Frontalis	19	19	18	18	17	16	17	16
Orbicularis palpebrarum	19	20	21	19	19	18	18	17
Pyramidalis nasi	19	19	18	18	21	19	19	18
Levator labii superioris alæque nasi	18	18	18	18	18	15	17	19
Triangularis nasi	19	19	18	17	19	16	17	18
Orbicularis oris	19	19	19	18	17	16	18	19
Zygomaticus major	17	17	17	17	16	14	14	16
Depressor anguli oris	18	18	18	17	19	16	16	16
Platysma myoides	18	19	18	17	20	18	19	18

TABLE II.

Instrument used = smaller apparatus.

Name of muscle	W. F. 1st sitting	Do. 2nd sitting
Sterno-mastoid	19	19
Trapezius, upper and external portion	20	...
,, lower portion	17	17
Lattisimus dorsi	17	16
Pectoralis major	17	18
Deltoid	20	21
Biceps flexor cubiti	17	16
Flexor sublimis digitorum	16	17
Extensor communis ,,	15	16
Interossei (dorsal)	16	16

TABLE III.

Instrument used = smaller apparatus.

Name of muscle	T. D.
Orbicularis oris	20
Triangularis nasi	18
Levator labii superioris alæque nasi	18
Orbicularis palpebrarum	23
Levator alæ nasi	20
Frontalis	19
Biceps flexor cubiti	19
Flexor sublimis digitorum	20
Extensor communis ,,	18

It will be seen upon examining these tables, that amongst ordinarily healthy men, there are variations with regard to the electro-contractility of the muscles ; and that there are also differences in this respect in the same man at different times. These are not so well marked, however, as those between the muscles of separate individuals. Although several days elapsed between the different sittings of W.F., yet there is considerable uniformity in the results; indeed, in some of the muscles, the power required to produce contraction was exactly the same on all four occasions. On comparing these results with those of the faradisation of the muscles in the case of M.B., wider disparities are observed ; and in the latter case there is not the same uniformity between the numbers on the separate days of examination as in the former. The variations in the case of M.B. are also greater in the power required to produce contraction of individual muscles. Table II. gives the results of the examination of some of the muscles of the limbs and trunk of a healthy man on two occasions. It shows that the upper and external portion of the trapezius muscle is more excitable than its lower portion. Duchenne points out this, and attributes it to the fact that the former portion of the muscle receives branches from the external division of the spinal accessory nerve. The deltoid muscle, as the table shows, in this case equalled the upper part of the trapezius as regards excitability. Table III. gives the results of the examination of several muscles of another sane man in ordinary health. Several of the muscles in this case were clearly more excitable than in the former instances. From these experiments on healthy men the following conclusions may be drawn:—

(1). That the electro-muscular contractility varies in different individuals in ordinary health.

(2). That the muscles of the face are generally more excitable than those of the limbs and trunk.

(3). That different portions of the same muscle may vary as regards electro-contractility.

(4). That variations occur in the electro-contractility of the same individual at different times.

2. *General paralysis of the insane.*—From the defective articulation, the tremor of the labial and other facial

muscles, and the want of control over the limbs, as exemplified in the defective handwriting and altered gait in general paralysis of the insane, it is a disease in which we should *à priori* expect to find great changes as regards the electro-irritability of the muscles. An examination of Table IV. shows that in the first and second stages of the disease, there is neither exalted nor diminished electro-excitability of the muscles of the face.

TABLE IV.

Instrument used = smaller apparatus.

Name of muscle	J. H.	H. P.	T. T.	C. W.	R. A.	J. W.
Frontalis	18	18	18	18	15	17
Orbicularis palpebrarum	19	19	20	18	16	20
Pyramidalis nasi	19	19	17	19	17	16
Levator labii superioris alæque nasi	19	19	19	21	17	19
Triangularis nasi	18	18	17	18	17	17
Orbicularis oris	20	20	18	19	19	18
Zygomaticus major	18	16	16	16	16	16
Depressor anguli oris	19	18	18	17	18	18
Levator menti	18	18	...	17	17	18
Quadratus menti	18	18	...	17	18	18
Platysma myoides	19	18	18	18	17	17

Table V. gives the results of the examination of several of the facial muscles, and of some of the muscles of the arms and legs of seven general paralytics who were all in the last

TABLE V.

Instrument used = larger apparatus.

Name of muscle	E. D.		G. B.		T. P.		R. H.		H. T.		W. N.		C. W.	
	R.	L.	R.	L.	R.	L.	R.	L.	R.	L.	R.	L.	R.	L.
Frontalis	13	12	13	14	..	..	12	12	..	..	14	..	13	10
Orbicularis palpebrarum	..	..	12	12	..	..	..	..	..	..	12	..	11	10
Levator labii superioris alæque nasi	14	15	14	13	..	..	11	11	..	..	..	..	12	11
Zygomaticus major	11	13	10	10	..	..	11	11	..	..	12	8	10	9
Depressor anguli oris	11	13	10	10	..	..	9	9	..	..	12	..	10	10
Platysma myoides	13	13	14	13	..	..	13	13	..	..	..	..	13	14
Biceps flexor cubiti	10	12	10	12	5	5	13	13	13	13	13	..	9	12
Extensor communis digitorum	10	10	3	10	5	5	7	7	9	8	..	..	8	10
Gastrocnemius	..	..	..	..	5	5	7	7	8	8	9	..	1	1
Tibialis anticus	..	..	..	..	5	5	n.	n.	n.	n.	9	..	1	1

stage of the disease. They were inmates of the West Riding Asylum, and the observations were made with

Stöhrer's larger apparatus. In these seven cases, the corresponding muscles of opposite sides of the body were subjected to experiment separately, in order to ascertain whether the muscles of opposite sides presented differences as regards their electro-excitability.

A glance at the table shows that, generally speaking, there was great similarity in this respect. In some instances there was diminished irritability of the muscles of the arms. This was the case in the muscles of the legs in all the instances in which they were examined ; and, in two cases, no contraction of the tibiales antici could be produced by the highest power of the secondary current. It is a noteworthy fact that in these seven cases the power of locomotion was impaired or altogether wanting.

TABLE VI.

Instrument used=smaller apparatus.

Name of muscle	C. W.	F. W.	J. D.	J. C.	J. J. H.	J. G.	M. D.	J. R.	E. T.	R. H.	J. C.	W. L.	T. H.	S. W.	J. B.	A. P.	J. S.	S. A.	W. T.	W. S.	H. B.	E. H.
Frontalis	17	17	16	16	17	15	16	16	16	16	17	16	16	17	14	15	16	17	17	16	17	16
Orbicularis palpebrarum . . .	20	17	20	20	17	17	16	18	18	17	18	18	18	19	16	16	17	20	18	16	19	18
Pyramidalis nasi	20	18	17	18	18	17	15	17	17	16	18	19	18	18	16	17	17	20	19	17	18	17
Levator labii superioris alæque nasi	19	16	16	16	17	18	17	16	16	16	17	16	17	18	16	17	19	19	23	18	17	18
Triangularis nasi	20	17	15	17	16	17	18	15	16	16	18	16	17	19	14	16	18	19	18	16	18	18
Orbicularis oris	18	20	18	17	21	18	18	19	19	17	18	19	19	20	17	17	18	18	18	18	17	19
Zygomaticus major . . .	18	18	17	15	16	16	16	16	15	15	15	14	16	16	13	15	16	18	17	14	17	17
Depressor anguli oris . . .	17	17	18	17	17	17	17	16	16	16	17	16	16	18	14	16	16	18	16	15	17	17
Levator menti	17	18	15	17	18	16	17	17	15	16	17	17	17	18	17	16	16	17	17	16	17	17
Quadratus menti	17	18	17	17	18	18	17	17	16	16	17	18	17	18	16	17	16	18	18	15	18	17
Platysma myoides . . .	17	18	20	16	19	19	18	18	17	17	16	17	17	19	17	16	17	17	18	16	18	20

3. *Epilepsy.*—As the above table speaks for itself, little need be said on this subject. The results given in the table are very much like those obtained in healthy men. None of these cases were in the *status epilepticus* at the time the observations were made, so that I cannot say what the state of the muscles may be in that condition ; but I have no hesitation in saying that, otherwise, there is nothing worthy of note with regard to the electro-excitability of the facial muscles amongst epileptics.

4. *Mania.*—From the results of the examination of three cases of mania, which are given in the accompanying table, it will be seen that here also there is nothing deserving of special comment. The first two cases in the table were in a

state of chronic excitement. The third was that of a man who is generally silent and sullen, and in the habit of standing all day in one position, but who at times makes sudden attacks upon the attendants.

TABLE VII.

Instrument used=smaller apparatus.

Name of muscle	E. B.	H. W.	W. E.
Frontalis	16	18	18
Orbicularis palpebrarum	18	18	17
Pyramidalis nasi	21	19	18
Levator labii superioris alæque nasi	20	19	16
Triangularis nasi	19	19	18
Orbicularis oris	18	18	18
Zygomaticus major	16	15	15
Depressor anguli oris	16	17	15
Levator menti	17	18	17
Quadratus menti	17	19	17
Platysma myoides	19	20	21

5. *Dementia.*—The following table gives the results of the examination of the facial muscles of two patients in this asylum labouring under chronic dementia.

TABLE VIII.

Instrument used=smaller apparatus.

Name of muscle	J. H.	G. G.
Frontalis	18	17
Orbicularis palpebrarum	20	19
Pyramidalis nasi	19	19
Levator labii superioris alæque nasi	16	16
Triangularis nasi	18	17
Orbicularis oris	17	17
Zygomaticus major	15	16
Depressor anguli oris	16	16
Levator menti	17	15
Quadratus menti	17	19
Platysma myoides	18	18

About these there is nothing unusual to call for remark. At the West Riding Asylum, Wakefield, I met with two typical examples of acute dementia. In one of these, the sensibility of the skin was so great that, after the electrodes had once touched the patient's face, he was so terrified that he

became quite energetic in his endeavours to avoid them. In the other case, the facial muscles readily responded to the electric stimulus, and I had some photographs taken illustrating the action of several of them under it. The contrast was most marked between the ordinary dull and unconcerned expression and the broad grin produced by the contraction of the zygomatic muscles. The electro-cutaneous sensibility in this case appeared to be normal.

6. *Chronic disorganisation of the brain.*—The results in Table IX. were obtained from the examination of the muscles of a patient in this asylum, who is a typical example of

TABLE IX.

Instrument used = smaller apparatus.

Name of muscle	W. McM.
Frontalis .	15
Orbicularis palpebrarum .	16
Pyramidalis nasi .	16
Levator labii superioris alæque nasi .	16
Triangularis nasi .	16
Orbicularis oris .	17
Zygomaticus major .	15
Depressor anguli oris .	15
Levator menti .	17
Quadratus menti .	15
Platysma myoides .	18
Gastrocnemii .	8
Tibiales antici .	1

chronic disorganisation of the brain of long standing. This patient had great difficulty in walking, being almost paraplegic. The contractility of the gastrocnemii was impaired, and the tibiales antici required the full force of the current to cause their contraction.

Table X. gives the results of the faradisation of the muscles in a case of chronic disorganisation of the brain (alcoholic), the patient being an inmate of the West Riding Asylum. The instrument used was Stöhrer's larger induction apparatus. In this case there was impaired excitability of the zygomatic muscles, and of the extensor communis digitorum; and no contraction of the gastrocnemii could be produced, or of the tibialis anticus of the left side, although the same muscle of

the right side contracted readily enough. I could find no explanation of this difference.

TABLE X.

Name of muscle	T. T.	
	R.	L.
Frontalis	12	12
Orbicularis palpebrarum	12	12
Levator labii superioris alæque nasi . .	13	13
Zygomaticus major	5	5
Depressor anguli oris	...	...
Platysma myoides	10	10
Biceps flexor cubiti	9	9
Extensor communis digitorum . . .	1	1
Gastrocnemius	*n.*	*n.*
Tibialis anticus	11	*n.*

7. *Progressive muscular atrophy* :—I have found that, in cases of this disease, the contractility is impaired in the affected muscles, and I believe with Dr. Althaus that ' farado-muscular contractility is, in this disease, quite proportional to the more or less atrophic condition of the fibres.' [1]

The following case may be given as an example :

The patient was a congenital idiot, and was a patient in the West Riding Asylum. The following is an extract from the case-book describing the condition of his muscles when admitted, and applies to him as he was when I examined him :—' The legs are in a flexed position, due to contraction of the flexor tendons. . . . All the muscles of the body are wasted, especially those of the legs and arms, and the toes and fingers are drawn in and contracted. . . . There is now great wasting of the muscles of the legs —especially the gastrocnemii.' I faradised some of the muscles of the legs and arms with the result given in Table XI.

The muscles of the arm, in which the wasting was slight, contracted readily. In the muscles of the legs, on the other hand, in which extensive atrophy and degeneration had taken place, no contraction could be produced.

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[1] A treatise on ' Medical Electricity,' by Julius Althaus, M.D., p. 425.

TABLE XI.

Instrument used = larger apparatus.

Name of muscle	R. E.	
	R.	L.
Biceps flexor cubiti	13	13
Extensor communis digitorum . . .	10	11
Gastrocnemius	*n.*	*n.*
Tibialis anticus	*n.*	*n.*

8. *Hemiplegia.*—Dr. Marshall Hall first directed attention to the value of electricity in distinguishing between cerebral and spinal paralysis. He asserted that in cerebral paralysis the irritability of the muscles was increased, and that in spinal paralysis it was diminished or altogether wanting. Dr. Todd found that in cerebral paralysis, in one class of cases the irritability of the paralysed muscles was increased; that in another it was diminished; and that in a third class it was the same in the paralysed as in the healthy muscles. Duchenne found that muscular contractility was normal in cerebral paralysis. Dr. Althaus has 'tested the nervous and muscular excitability in more than a hundred cases of cerebral paralysis,'[1] and agrees with Dr. Todd.

The following account of a case of cerebral paralysis, which I examined at the West Riding Asylum, Wakefield, may be of some interest :—W. B—, æt. 66, suffering from chronic atrophy of the brain, was admitted to the West Riding Asylum on October 28th, 1872. There was a history of drunkenness. The following notes are extracted from the case-book : ' 1873. January 26th. Little or no change. April 18th : Had another paralytic stroke yesterday, affecting the left side, and more severe than the previous ones have been. April 21st : There is distinct drawing of the mouth to the affected (left) side. He is quite unconscious to-day. Breathing stertorous. Pupils contracted. When the left arm is raised it falls heavily. This is not so well marked on the right side. Reflex sensibility is still retained in the lower extremities.

[1] A treatise on ' Medical Electricity,' by Julius Althaus, M.D., p. 398.

Pulse 126. . . . .' Whilst he was in the condition described in the last note, I tested the electro-excitability of some of his muscles, with the results given in Table XII.

TABLE XII.

Instrument used=larger apparatus.

Name of muscle	W. B.	
	R.	L.
Orbicularis palpebrarum	11	11
Levator labii superioris alæque nasi . .	8	8
Zygomaticus major	6	6
Depressor anguli oris	11	12
Biceps flexor cubiti	15	14
Extensor communis digitorum . . .	10	*n.*
Tibialis anticus	*n.*	*n.*

The result here is somewhat singular, viz., that the biceps contracted in each arm with about the same strength of current ; whereas the extensor communis digitorum of the paralysed arm would not contract at all, although that of the other arm responded readily. No contraction could be produced in the tibialis anticus of either leg. The electro-contractility was the same in the muscles of the two sides of the face, notwithstanding the fact that there was flattening of the features of the *right* side of the face, the muscles of that side being paralysed, and the corner of the mouth being drawn to the left side. Reflex electro-sensibility was greater on the *right* side of the face than on the *left*, and in the *left* leg than in the *right*. The patient expired soon afterwards, and, at the post-mortem examination, the following were the appearances found in the brain : (I quote from the post-mortem book) 'There is slight wasting of the gyri of the frontal and parietal lobes. The ventricles are full of clear serum, and their lining membrane is granular. There is a small softened point about the size of a pea at the anterior margin of the right corpus striatum.'

9. *Locomotor Ataxy.*—Table XIII. gives the results of the examination of the muscles of two male patients in the West Riding Asylum, both of whom had long suffered from

locomotor ataxy in addition to their mental disorder. W. M. showed quiet mental exhilaration and self-satisfaction. He was very stupid and considerably demented. The locomotor ataxy had so far advanced, that he was perfectly helpless, and unable to walk. T. P. suffered from melancholia. He was fretful and irritable, at times, when the neuralgic pains troubled him. He was equally helpless with W. M., having quite lost his locomotive powers. I tested a number of the muscles in these two cases with Stöhrer's larger instrument, and the result is seen in the table.

TABLE XIII.

Instrument used = larger apparatus.

Name of muscle	W. M.	T. P.
Frontalis	13	13
Orbicularis palpebrarum	14	12
Pyramidalis nasi	16	14
Levator labii superioris alæque nasi . .	16	14
Triangularis nasi	15	12
Orbicularis oris	15	13
Zygomaticus major	13	9
Depressor auguli oris	13	11
Levator menti	14	11
Quadratus menti	14	10
Platysma myoides	14	11
Biceps flexor cubiti	14	11
Gastrocnemius	n.	n.

The contractility was, as the table shows, more easily excited in W. M. than in T. P., in all the muscles with the exception of the frontalis. In neither case could contraction of the gastrocnemii be produced. While residing at the West Riding Asylum, I had the good fortune to meet with two very well-marked cases of locomotor ataxy in sane men, both of whom very kindly allowed me to test the electro-contractility of their muscles. One of these resided in the immediate neighbourhood of the asylum, the other was the husband of a female patient in the asylum, who was in the last stage of general paralysis of the insane. I will briefly describe these cases, and the effect of the faradic current upon their limbs.

CASE 1.—J. W., æt. 37, widower. Never had any children. *History.*— Was a ' stuff-presser ' by trade, and was exposed to great changes of temperature. The symptoms of his present disorder began about six years ago. Until that time, he had always enjoyed good health. *Present condition.*— He is stoutly built, has a healthy appearance, and is about the average height. States that his memory is as good as it ever was, and that his eyesight is not at all impaired. He cannot stand in the dark or with his eyes shut; and cannot walk without the aid of crutches and some one to assist him. When lying in the recumbent posture, he has perfect control over his legs, can move them about at will in any direction, and, when his legs are flexed, can offer powerful resistance to anything placed against the soles of his feet; but he cannot tell whereabouts his feet are, when he is in bed, unless he sees them. The tips of his fingers ' feel thick,' and he cannot button anything with them, unless he sees what he is about.

On applying the highest power of the secondary current of Stöhrer's larger apparatus to the gastrocnemii and tibiales antici, no contraction of these muscles takes place. Except at one spot, over the gastrocnemius, he feels nothing during the application but the sensation of cold produced by the moistened electrodes. On applying the current of the *primary* coil to the same muscles, no contraction results, but, on placing the electrodes over the abductor pollicis pedis, contraction of the latter muscle at once takes place. The condition is the same in both legs. He states that he experiences a pricking sensation during the application of the current of the primary coil. On applying the current of the secondary coil to his arms, he feels it at once, and the muscles respond readily.

CASE 2.—W. L., æt. 43, sculptor, Leeds. Married, but never had any children. This patient was examined by Dr. Burman, of the West Riding Asylum, on October 29th, 1872, and the following account of his condition is from notes taken by Dr. Burman at that time, he having kindly allowed me to make use of them. ' The affection of the legs commenced about five years ago, and the onset was gradual. The great symptoms were aching pains when going upstairs, and after that he got worse, and got nervous and queer; but for the last two or three years he has been in a stationary condition, being just able to walk about slowly with a stick. *Present condition.*—Stands well with feet spread out, and cannot stand with the eyes shut. Cannot walk without a stick, or in the dark. He is a sculptor, and can still work, and does work, but the hands are numb. Feet are very numb, and, when walking in the streets, he can scarcely tell that he is walking at all: his feet feel soft underneath. Lifts up his feet, and stamps when walking. Sensibility of the skin is much more acute than it used to be in the upper part of the body. The division between the two is well defined. Examination of legs: No movements on tickling the feet. He cannot feel tickling at all. . . . Feels a prick with a pin if roughly done. . . . He feels it some time after the prick, and it is not at all uncomfortable. Sensibility of the dorsum of the foot impaired in like manner, though it is not so dead as the sole. A strong interrupted current—the poles applied at the soles of the feet . . . . has no effect on him, and he does not feel it. The muscles of the legs, however, readily respond to the electric stimulus, and

contract, but even then he scarcely feels the current. When walking without boots, he feels as if he had a brick tied to each foot.'

When I saw this patient, about six months after the above notes were taken, he was in very much the same condition as at that time. The disease appeared to have made no further progress. Indeed, 'being just able to walk about slowly with a stick' scarcely applied to him then, for I saw him walking past the asylum, stick in hand, with considerable alacrity. On applying the current of the secondary coil of Stöhrer's larger apparatus to the tibiales antici and gastrocnemii, contraction of these muscles readily took place, but he stated that he felt nothing but a sensation of cold at the points where the electrodes were applied. The biceps and extensor communis digitorum contracted readily in both arms. He stated that if he scratched his legs violently, he had to continue doing so for some time before he could feel it. This peculiarity was most marked in the legs, especially below the knee; but existed also in the arms.

10. *Melancholia.*—In one or two cases of hypochondriacal melancholia, which I examined at the West Riding Asylum, there was nothing specially noteworthy. In a case of recurrent melancholia, the subject of which had long entertained the delusion that he could not use his legs, and was, consequently, wheeled about in a chair, the muscles of the legs, having undergone atrophy and degeneration from want of use, gave no response to the faradic stimulus. No paralysis of his legs existed, except in his imagination.

To sum up, then : in cases of general paralysis, chronic disorganisation of the brain, and locomotor ataxy, in which the power of locomotion was affected to such an extent as to prevent the patient walking without assistance, the electro-contractility of the muscles of the legs was impaired or altogether gone.

The muscles of the face and arms, in these cases, responded readily to the electric stimulus. In progressive muscular atrophy, the electro-muscular contractility was diminished according to the extent of the disease. Bearing in mind the physiological law, that an organ whose function has for some time lain dormant undergoes atrophy and degeneration, I think that wasting of the muscles and degeneration of their substance, although not sufficiently marked to be apparent, may in a great measure explain their refusal to contract under the electric stimulus in the cases of brain-wasting, general paralysis, and locomotor ataxy before mentioned.

Take, for example, the two cases I have recorded of locomotor ataxy occurring in sane men. In one of these, the disease had not progressed sufficiently to prevent the patient walking readily enough with the aid of a stick: in the other, it had advanced so far that the patient could make no attempt at locomotion without assistance, and, consequently, the muscles of his legs were seldom brought into action. In the latter case, the muscles scarcely responded at all to the current; in the former, their electro-contractility was normal. The case of recurrent melancholia, referred to above, also bears out this view.

I need say nothing further regarding the case of cerebral hemiplegia which I have before recorded. From the results of the examination of the cases of epilepsy, mania, and dementia, and of the remaining cases of general paralysis and melancholia, it may be said that, in these diseases, in their earlier stages there is no change as regards the electro-contractility of the muscles.

Electro-Excitability of the Sensibility *of Muscles, and of the Skin.*—I have not made a special series of observations with regard to this subject, but have noticed, incidentally, some striking facts regarding it.

Both the electro-cutaneous and electro-muscular sensibility seems to be increased in paralysed limbs. It was so in the case of cerebral hemiplegia already recorded. The following case is another example: ' H. O., æt. 71. Left arm and leg paralysed for fifty years. There is great wasting of the muscles of the affected leg. He feels the current more in that leg, and in the affected arm, than in the sound limbs.'

In several cases, I have found extreme sensitiveness of the face to the action of the current. In the cases of locomotor ataxy recorded above, the muscular and cutaneous sensibility of the legs to the action of the secondary current was quite gone. This was the case in several other instances, amongst which may be mentioned that of an imbecile, whose locomotive power was much impaired. I hope to have the opportunity of recording, at some future time, some experiments upon this subject.

HEART DISEASE AND INSANITY.

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Since the time that melancholy was vaguely attributed, by the ancients, to an excess of black bile in the blood, mental insanity has ever been considered to stand in important relation, as to many of its varieties, to various bodily diseases,[1] and, conversely, a recognition of the influence of mind on the bodily functions is of quite as old a date. It is only of late years, however, that we have arrived at any thorough appreciation of the mutual inter-dependence of body and mind, and been able to apply our knowledge and experience of it to practical purposes, and thus reap rich results from our more rational, and, consequently, more beneficial treatment of cases of insanity. We are much indebted for our increased knowledge of this mutual dependence of body and mind, to Dr. Maudsley, on the one hand, who, in his very able and interesting Gulstonian Lectures for 1870, surveys the whole question of the connection and mutual influence of body and mind, from a general point of view, and to Dr. Daniel H. Tuke, on the other, who, in a series of papers in the 'Journal of Mental Science,' commencing in July, 1870, treats more particularly of the influence of the mind upon the body. I think I shall be within bounds, when I venture to assert, that the great primary indication for treatment, in cases of insanity, now-a-days, is

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[1] I use the term '*bodily* diseases' here for convenience' sake, and it must not, of course, be understood to include organic diseases of the brain itself.

to aim at removing bodily disorder, and curing bodily disease, if such exist ; and it is well known that this treatment is, of itself, often sufficient to restore mental health and vigour.

Besides this intimate relation of mental diseases to certain general bodily diseases and disorders of nutrition, the connection of certain special forms of insanity with various particular diseases of the bodily organs has now, furthermore, been so clearly established as to justify the use of the term ' *sympathetic*,' as applied, by Van der Kölk and other continental writers, to the varieties of insanity included under this category. The sympathies of the brain, for instance, with the generative organs, in both sexes, and with various parts of the intestinal tract, cause disorders and affections of the latter, with their concurrent results, to react on the brain, so that well-marked forms and modifications of insanity are produced, the only radical treatment for which is the removal or amelioration of the bodily disorder.

The connection between bodily and mental diseases being often, then, of such an intimate character, it necessarily follows that a more extended and special investigation into the relations which exist between insanity and some of the most common bodily diseases, should tend to the elucidation of many facts which cannot fail to be of great value to the physician-alienist. Such special investigation, with regard to phthisis—that bodily disease which, as a cause of death, claims, annually, a greater number of victims than any other in our country, and which is a cause of death in about 15 per cent. of asylum cases—has already been undertaken, and conducted in his usual careful manner, by Dr. Clouston,[1] who has shown, *inter alia*, that, in those cases in which the development of phthisis and insanity has been nearly contemporaneous, the mental symptoms are sufficiently peculiar and uniform to justify his placing them in a natural group, under the designation of *phthisical mania*.

With regard to heart disease, which ranks eighth, in order of merit, as a cause of death, according to the last report of

[1] ' Journal of Mental Science,' April 1863.

the Registrar-General, and which seems to be steadily increasing in frequency year by year, I am not aware that any special investigation has been made as to the relation in which it stands to insanity ; and yet, considering the intimate and peculiar sympathy which exists between the heart and the brain, and their special mutual dependence and relations to one another, it would seem that there are *à priori* reasons for supposing that such an investigation would not be altogether barren of good result. At any rate, I have ventured to make some little enquiry into the matter, and, without any attempt at proving foregone conclusions, I shall impartially record the result of my observations.

I shall first endeavour to solve the problem as to whether or not such statistics, as we can avail ourselves of, show any general causal relation between heart disease and insanity. And, in order to establish any such relation, it would, at least, require to be demonstrated—*firstly*, that the two diseases are increasing in frequency *pari passu* ; *secondly*, that those parts of England and Wales in which there is—presumably from the greater mortality from it—a larger proportion of cases of heart disease, should yield a larger proportion of insane people than other parts in which, *cæteris paribus*, heart disease is less frequent, and *vice versâ*, we should get the opposite result; and, *thirdly*, that heart disease is more frequent in asylums than out of them.

Let us now proceed to investigate these three points separately :—

I. That there is a steady increase, annually, in the numbers of the registered insane, both absolutely and proportionately to the increase in general population, Table No. I[1] clearly shows ; as well as that the *proportionate* increase is by no means of an alarming nature. The population for each year, in the table, has been taken from the Annual Reports of the Registrar-General, and is applicable to the middle of each year.

And that there is a similar steady increase in the numbers

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[1] Taken from page 5 of the twenty-sixth Annual Report of the Commissioners in Lunacy.

TABLE I.

Showing the Ratio per 1,000 of the Total Number of Lunatics, Idiots, and Persons of Unsound Mind, to the Population, in each Year, from 1859–1872, both inclusive.

Year	Population	Total Number of Lunatics, Idiots, &c., on 1st January	Ratio per 1,000 to the Population
1859	19,686,701	36,762	1·86
1860	19,902,713	38,058	1·91
1861	20,119,314	39,647	1·97
1862	20,336,467	41,129	2·02
1863	20,554,137	43,118	2·09
1864	20,772,308	44,795	2·15
1865	20,990,946	45,950	2·18
1866	21,210,020	47,648	2·24
1867	21,429,508	49,086	2·29
1868	21,649,377	51,000	2·35
1869	21,869,607	53,177	2·43
1870	22,090,163	54,713	2·47
1871	22,704,108	56,755	2·49
1872	23,074,600	58,640	2·54

TABLE II.

Showing the Deaths and Death-rate per 1,000 from Heart Disease in the Four Quinquennial Periods between 1851 and 1870.[1]

MALES

Quinquennial Periods	Deaths at different Ages					Annual Rate per 1,000 Living				
	All Ages	0–20	20–45	45–65	65 and upwards	All Ages	0–20	20–45	45–65	65 and upwards
1851–1855	32,617	4,416	6,454	11,873	9,874	·725	·177	·553	1·829	·507
1856–1860	39,678	3,433	8,723	14,629	12,893	·836	·156	·529	2·137	·627
1861–1865	49,738	3,942	11,128	18,662	16,006	·993	·169	·605	2·583	·738
1866–1870	57,687	4,296	13,089	21,132	19,170	1·085	·174	·709	2·757	·833

FEMALES

Quinquennial Periods	Deaths at different Ages					Annual Rate per 1,000 Living				
	All Ages	0–20	20–45	45–65	65 and upwards	All Ages	0–20	20–45	45–65	65 and upwards
1851–1855	33,497	4,784	6,719	12,165	9,829	·713	·171	·530	1·780	·423
1856–1860	41,095	3,610	9,349	14,930	13,206	·825	·164	·516	2·061	·536
1861–1865	51,243	4,153	11,025	18,897	17,168	·970	·178	·573	2·454	·657
1866–1870	60,003	4,632	12,711	21,588	21,072	1·067	·187	·621	2·632	·758

[1] Taken from page 440 of the thirty-third Annual Report of the Registrar-General.

of deaths from heart disease, both absolutely and proportionately, is equally evident from a consideration of Table II., which is a very valuable and interesting one, and of which I shall have occasion to make further use in the course of this paper. From a consideration of the Tables of Causes of Death in England, in the Registrar-General's Reports for the five years 1866–70, I have, furthermore, ascertained that the percentage of deaths, at all ages, from heart disease (excluding aneurisms) on the total deaths, has increased from 4·34 to 4·91; and, excluding deaths before the age of 20 (during which period heart disease is only a rare cause of death), in order to render the comparison, so far as an asylum population is concerned, more correct (the amount of insanity existing before the age of 20 being insignificant for present purposes), the percentage has steadily increased from 8·14 to 9·82.

With regard to this steady increase of deaths from heart disease, the Registrar-General, in his thirty-third Annual Report, p. 405, makes the following valuable remarks: ' The diseases of particular organs, which may be called *monorganic*, constitute the class of local diseases, to which, in the year 1870, 205,264 deaths were referred; the mortality was somewhat above the average by these increasing diseases. This is especially the case with heart disease, to which were referred 11,356 deaths in the year 1850, 18,758 deaths in 1860, and 25,259 in 1870. The increase was rapid and progressive in the twenty years.' He thinks it only fair, however, to assume that a *part* of the increase of heart disease in England is apparent rather than real, and due to improved nomenclature and advancing diagnosis—what was formerly called dropsy being now, more correctly, designated disease of the heart, and so on. He founds this assumption on the fact that, simultaneously with the increase of death by heart disease, there has been a decrease of deaths ascribed to dropsy. The Registrar-General only refers to this disturbing element in a general manner, so that I cannot ascertain in how far he considers it a source of fallacy; but Dr. Quain in his recent Lumleian Lectures, making all due allowance for this element, holds that there has been an

actual increase of heart disease in England, within the last twenty years.

On the whole, therefore, with regard to this point, I think it is clear enough that there has been something more than a *pari passu* increase of heart disease, as compared with mental insanity in England; and whilst it would be difficult to determine the exact significance of this relation of heart disease to insanity, as well as of the other relations of these diseases, the one to the other, as I have ascertained them to exist, and shall further illustrate, yet I venture to think the facts are not uninteresting, and worthy of record.

II. Let us pass, now, to a consideration of the next point, as to a probable causal relation between heart disease and insanity, to which I have referred, viz., II., including a comparison of the local distribution, throughout England and Wales, of cases of mental insanity, with that of the deaths from heart disease. But, I may, perhaps, just pause for a moment here, in order to express my surprise that, in his recent paper [1] on the local distribution of insanity and its varieties in England and Wales, Dr. Clouston, though he mentions phthisis, does not include a consideration of the connection which might exist between the local distribution of heart disease and insanity, in his comprehensive *schema* for the continuation of his most interesting and valuable paper, more particularly with a view to explaining the differences and peculiarities that exist in the local distribution of insanity and its varieties. Well, to return: I may state that my observations on this point are founded on a comparison of the local distribution of deaths from heart disease, for the ten years 1851–60—as shown in Mr. Haviland's monograph on the subject, and illustrated by the very excellent coloured chart therewith published—with that of the local distribution of insanity as it existed on January 1, 1871, which latter I have also illustrated by means of a coloured chart, used by Dr. Crichton Browne in his class-room, and kindly lent me by him. The table on which this chart of insanity is

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[1] 'Journal of Mental Science,' April 1873.

founded was published at p. 14 of the twenty-fifth Annual Report of the Commissioners in Lunacy, the population being put down according to the *estimates* of the Registrar-General; but Dr. Clouston, in the paper already referred to, has wisely corrected it in accordance with the *actual* census returns for that year subsequently issued, and thus republished it. For purposes both of convenience and reference, I must reproduce this table, as carefully revised by Dr. Clouston, and, in order to facilitate comparison with Mr. Haviland's results as to the distribution of heart disease, I have arranged the counties, not in alphabetical order, but in two groups, and have further subdivided them as they are found arranged together in the eleven registration divisions of England and Wales. This table, then, is an interesting one, and important to me for purposes of illustration, showing as it does the ratio per 1,000 of the total number of the registered insane, to the population in the various counties of England and Wales on January 1, 1871.

Let us now proceed to compare the local distribution of heart disease, as given and illustrated by Mr. Haviland, with the local distribution of insanity as shown in the above table. Those registration divisions which are, according to him, unhealthy (so far, at least, as regards heart disease), *i.e.* in which the mortality from heart disease is above the mean annual average rate of mortality for every 10,000 persons living, I have placed together in Group I., whilst Group II. contains those divisions in which the mortality from heart disease is below the average, and which may therefore be termed healthy—healthy *quoad* heart disease. Calculating the ratios on the aggregate totals in each Group, it will be found that the proportion of insane to the total population is greater in the first than the second group, being 4·34 per 1,000 in the group of divisions of highest mortality from heart disease, and 4·14 per 1,000 in the group of lowest mortality; which represents, on calculation, no less than an actual excess, *cæteris paribus*, of about 2,046 insane persons to be accounted for, in some manner or other, in the group of divisions of highest mortality from heart disease, or 1 more insane person to every 5,000 of

TABLE III.

Registration Divisions	Counties	Population 1871	Total Lunatics 1871	Lunatics per 1,000 of Population
England and Wales		22,704,108	50,637	2·2
VI. West Midland	Gloncester . . .	534,320	1,492	2·8
	Hereford	125,364	414	3·3
	Salop	248,064	695	2·8
	Stafford	857,333	1,264	1·5
	Worcester . . .	338,848	1,088	3·2
	Warwick	633,902	1,486	2·3
II. South Eastern	Surrey	1,090,270	2,590	2·4
	Kent	847,507	1,864	2·2
	Sussex . . .	417,407	1,060	2·5
	Hampshire . . .	543,837	1,312	2·4
	Berkshire . . .	196,445	713	3·6
V. South Western	Wilts	257,202	806	3·1
	Dorset . . .	195,544	489	2·5
	Devon . . .	600,814	1,438	2·4
	Cornwall . . .	362,098	567	1·6
	Somerset . . .	463,412	1,272	2·7
III. South Midland	Herts . . .	192,725	516	2·7
	Bncks . . .	175,870	441	2·5
	Oxford . . .	177,956	556	3·1
	Northampton . .	243,896	622	2·5
	Hunts . . .	63,672	134	2·1
	Beds . . .	146,256	377	2·6
	Cambridge . . .	186,363	446	2·4
X. Northern Counties	Durham . . .	685,045	893	1·3
	Northumberland .	386,959	773	2·
	Cumberland . .	220,245	463	2·1
	Westmoreland . .	65,005	131	2·
GROUP I.	Total	10,229,359	23,902	2·34
I. London	Middlesex . . .	2,538,882	7,312	2·9
VII. North Midland	Leicester . . .	268,764	805	3·
	Rutland . . .	22,070	51	2·3
	Lincoln . . .	436,163	867	2·
	Nottingham . .	319,956	786	2·5
	Derhy . .	380,538	597	1·6
IX. Yorkshire	Yorkshire . . .	2,436,113	3,662	1·5
IV. Eastern Counties	Essex . . .	466,427	1,017	2·2
	Norfolk . . .	438,511	1,135	2·6
	Suffolk . . .	348,479	853	2·4
VIII. North Western	Cheshire . . .	561,131	959	1·7
	Lancashire . . .	2,818,904	5,538	2·
XI. Monmonthshire and Wales	Monmouth . . .	195,391	547	2·7
	Anglesey . . .	50,919	91	1·8
	Brecon . . .	59,904	147	2·5
	Cardigan . . .	73,488	158	2·1
	Carmarthen . .	116,944	309	2·7
	Carnarvon . . .	106,122	261	2·4
	Denbigh . . .	104,266	147	1·4
	Flint . . .	76,245	202	2·8
	Glamorgan . . .	396,010	685	1·7
	Merioneth . . .	74,369	108	2·3
	Montgomery . .	67,789	197 .	2·9
	Pembroke . . .	91,936	256	2·8
	Radnor . . .	25,428	45	1·8
GROUP II.	Total	12,474,749	26,735	2·14

the population. This excess is not certainly very striking;
but is still, I think, sufficiently decided to be worthy of note;
and I do not see why the comparative greater prevalence of
heart disease in these divisions should not be an element

worthy of notice in a consideration of the probable causes
of this excess of insanity. At any rate, I believe further
observation will show—as I believe my own results, hereafter
to be recorded, will justify the statement—that heart disease
has really more to do with the production of insanity than is
now commonly supposed. But let us pursue the investiga-
tion further still; and we shall find that the following
counties, in which the proportion of lunatics to the popula-
tion is more than 3 to 1,000, viz., Berks, Hereford, Oxford,
Wilts, and Worcester, are all in the divisions of highest
mortality from heart disease; whilst the following counties,
in which the proportion is under 2 in 1,000, viz., Anglesey,
Cheshire, Denbigh, Derby, Glamorgan, Radnor, Yorkshire,
Cornwall, Durham, and Stafford, are all, except the three
last, included in the divisions of lowest mortality from heart
disease; and, from this point of view, the relation between
the two forms of disease is more striking. Still further
progressing from the general to the particular, let us try
and ascertain if, in those counties in which the mortality
from heart disease is the very greatest, the proportion of
insane to the population is the very highest; and I think
we shall find that it is so. Mr. Haviland's large chart
illustrates the distribution of heart disease throughout
England and Wales as it occurs in the registration *districts*,
and this map cannot, therefore, be used by me for compari-
son, *au coup d'œil*, with my own, showing the distribution of
insanity throughout the *counties*; but, fortunately, he pre-
sents us with smaller maps, in his folio *brochure*, showing
the distribution of heart disease as it exists in the registra-
tion divisions, as well as in the counties. Comparing Havi-
land's map, then, showing the county distribution of heart
disease, with my own, showing the county distribution of
insanity, the places of maximum intensity of shade in each
are observed at a glance to be identical; or, to put it in
terms, the four counties — Berks, Hereford, Wilts, and
Worcester—in which there is the very highest mortality
from heart disease, are not only four of the six already
referred to, in which alone the proportion of lunatics to the
population is above 3 in 1,000, but have a greater proportion

of insane to the total population than the other two of the six, viz. Leicester and Oxford, which, whilst they are counties in which the deaths from heart disease are above the average, yet are second-rate counties, in this respect, compared with the four above mentioned. On the other hand, of those counties in which the death rate from heart disease is the very least, nine, viz., Hunts, Cornwall, Lincoln, Cheshire, Lancashire, Yorkshire, Durham, Radnor, and Denbigh, have a ratio of lunatics to the population less than that of the average throughout all England and Wales and (excepting Stafford) include all those counties which have the very lowest ratio per 1,000 of lunatics to the population, as will be seen on reference to the table; whilst the other seven counties coming under this category, viz., Beds, Cambridge, Suffolk, Norfolk, Rutland, Monmouth, Pembroke, and Flint, all have a ratio of lunatics to the population only very slightly in excess of the general average throughout England and Wales, and none of them reach the higher ratio of 3 to 1,000.

With regard, then, to the second of the points I am now investigating, viz., the comparative local distribution of heart disease and insanity, I think I have made it pretty evident that there is a very striking and remarkable relation between the two diseases. It would of course be very rash, on these general grounds, to infer that, because the relation does exist, it must therefore be causal, yet that the relation has some special significance can scarcely be doubted, and it should be an important element to take cognisance of in any attempt to explain the differences that exist in the local distribution of insanity and its chief varieties throughout England and Wales.

III. Let us now consider the third point to which I have referred, in the consideration of a probable general causal relation of heart disease to insanity, viz., the question as to whether or not heart disease is more common amongst the mentally insane than the sane. I must first, however, draw attention to the difficulties which lie across the way towards a full and proper investigation of this point. The easiest part of the investigation is, of course, the comparison of

heart disease as a cause of death, as it exists amongst the sane and the insane respectively; but it is evident that this could only reflect but a very limited amount of light on the point in question. It by no means necessarily follows— supposing that heart disease is to some extent, directly or indirectly, a cause of insanity—that it must, therefore, be more common as a *cause of death* amongst the insane; for heart disease, sufficient to cause or modify insanity (as I have reason to believe it can do), need not necessarily be rapidly fatal; it may not even be discoverable after death; it may be purely functional, or not sufficiently organic to be noticed, *post-mortem*, as a cause of death, in comparison with other causes. No one supposes that heart disease is a more severe and fatal disease amongst the mentally insane than the sane. We shall, however, give our results with regard to this as we have obtained them; but they must be taken *quanto valeant*. With respect to another part of this investi- gation, viz., the comparative frequency of heart disease amongst the sane and the insane, as it exists during life, whether functional or organic, and as found after death, to whatever extent, we certainly ought to find it more common amongst the insane than the sane, if, as we believe, heart disease stands in some causal relation to insanity. To ascertain this point we should require to examine equal and sufficient numbers of hearts of sane and insane people, both during life and after death, and compare the results: and the numbers would require to be pretty large in order to get a reasonable generalisation. This would be an immense labour, and scarcely within the scope of a paper on ' Heart Disease and Insanity,' even had I unlimited space at my dis- posal; so that I must content myself, in the meantime, with recording my observations with regard to the frequency of heart disease as it exists amongst the insane, deferring, for the present, a consideration of its *comparative* frequency. With these prefatory remarks as to the general difficulties in the way of solving the problem, as to whether heart disease is more common amongst the insane than the sane, let us now pass to consider—

Heart Disease as a Cause of Death amongst the Insane.

For purposes of comparison, I shall first record a few facts I have collated with regard to heart disease as a cause of death *generally.* According to Haviland, his remarks being founded on the Registrar-General's returns for the ten years 1851–60, more than one-seventeenth of the total mortality throughout England and Wales is attributed to heart disease and dropsy. The numbers of deaths from these causes are both absolutely and relatively greater amongst females than males—absolutely, inasmuch as 109,527 of the total number of cases were males, and 127,446, females; relatively, since there were 13·1 deaths of females from these causes, as compared with 11·8 of males, to every 10,000 persons living. The female mortality is less at the *extremes* of life than the male. Between the ages of 25 and 75, in both sexes, each decade has a relative mortality nearly double that of the one which precedes it. According to the Registrar-General's returns for the five years 1866–1870, I find that the mean annual average percentage of deaths from heart disease (excluding aneurisms) on the total deaths from all causes and at all ages, is 4·66. So much, then, in a general way, with regard to heart disease as a cause of death; and now for a consideration of it, as such, amongst the insane at the West Riding Asylum. Out of 2,476 consecutive deaths (males, 1,372; females, 1,104) from all causes, from January 1857 to March 1873, heart disease was certified as a cause of death in 184 cases, of which 100 were males, and 84 females; being 7·43 per cent. of the total number, or 7·28 of the males, and 7·60 of the females; the deaths ascribed to heart disease being thus greater, in proportion to the total deaths, in the West Riding Asylum than throughout England and Wales, as 7·43 per cent. is to 4·66. This would appear, at the first glance, to represent a very considerable excess of deaths from heart disease in the West Riding Asylum as compared with all England; and one would be inclined to attach a weighty significance to the fact, especially in a county, or rather part of a county, in which heart disease

is at a minimum as compared with the rest of the country. But this general difference is more apparent than real, and, if we are anxious to avoid rash and specious conclusions, as I trust we are, we must eliminate one great source of fallacy in this comparison. Strange to say, as I have ascertained from the last five annual reports of the Registrar-General, just about one *half* of the whole mortality throughout England occurs before the age of 20 ; and as Asylum deaths occur, for the most part, in persons *above* 20 years of age (only 13 out of the last 500 deaths in the West Riding Asylum being deaths of persons below that age), it is evident that, in order to make the comparison strictly accurate, we must calculate the percentage, not on the *total* deaths, but on about *half* of them. Having, then, made the necessary subtraction of the deaths under 20 years of age from the total deaths, I find that the percentage of the residuum which die of heart disease is a mean annual average one, for the last five years, of 8·58. Comparing this with 7·43, the percentage of West Riding Asylum deaths from heart disease, the difference is small, and the country, generally, shows an excess, as compared with the insane in West Yorkshire. The smaller fatality of heart disease in Yorkshire generally (though it contains one very black and isolated district, viz. the Wharfedale Union, in which the mortality from heart disease is very great), as compared with the rest of the country, will probably account for this slight difference, and render the percentages about equal. So far, then, as the West Riding insane may represent lunacy generally, it does not show an excessive *mortality* from heart disease, as compared with the population at large ; on the contrary, the mortality from it seems to be the same in the Asylum, as throughout the Riding which supplies it with patients, which is just the result one should expect to get, looking at the matter from an unbiassed point of view. The results would, no doubt, be different in those counties where the mortality from heart disease is high, and I should think that further information on this point might prove interesting, if one could only get it.

I pass on to further analyse the deaths ascribed to heart

disease in the West Riding Asylum. As throughout the country at large, it will be observed that it is a slightly, but still decidedly, more common cause of death amongst the females than the males. It was the *sole* cause of death in 52 out of the 184 cases—viz. 30 males and 22 females. In 40 other cases (m. 17, f. 23) it was certified, with others, as a primary cause of death; being in the remainder of the cases, of course, returned as a secondary cause. The heart disease was returned, as being associated with dropsy, in 13 cases (m. 7, f. 6); and whilst, in some of these, the heart disease, in others, the general dropsy, is returned as the primary cause of death, yet it would be difficult to give strict precedence to either one or the other, though I have just classified them as I have found them certified. Of the 184 cases, 24 (m. 17, f. 7) either died suddenly or were found dead in bed, Coroner's inquests being held in 16 of these cases. In only 1 case did *rupture* of the heart occur, and that was in the person of a male aged 72, the subject of melancholia, who died suddenly whilst on a night-stool. The form of mental disease, as certified on admission, was, in 68 cases, mania (in its various forms); in 56, dementia (including simple, senile, and epileptic dements); in 33, melancholia; in 18, dementia with excitement (principally cases of general paralysis); in 6, imbecility; in 1 case, *folie circulaire*; and in 2 cases the mental condition cannot be ascertained. Table IV. shows

TABLE IV.

Length of Residence			M.	F.	Total
3 months and under	.	.	32	25	57
3 to 6 months	.	.	10	5	15
6 to 12 ,,	.	.	9	7	16
1 to 5 years	.	.	22	20	42
5 to 10 ,,	.	.	11	12	23
Above 10 years	.	.	16	15	31
Total	.	.	100	84	184

the length of residence of the patients in the Asylum at the time of death; and from it, we may ascertain how grave a complication of mental insanity heart disease is—very nearly

one third of the cases, in which heart disease was certified as
a cause of death, living no longer than 3 months, and very
nearly one half dying within 12 months after admission.   In
these cases, we may certainly presume that the heart disease
existed on admission ; whilst, in the others, it may or may
not have been developed subsequently.

I shall arrange my further observations, as to the occur-
rence and nature of heart disease amongst the insane, under
two heads—viz. 1st, as it is found after death, and, 2nd, as it
is found to exist during life ; and, then, finally, we shall
essay a few remarks on heart disease,—1st, as a cause of, or
modifying agent in cases of, insanity ; and 2nd, as a result of
insanity.

Heart Disease, as found after Death, amongst the Insane.

My observations, under this head, are founded on the
records of the last 500 *post-mortem* examinations of patients
dying in the West Riding Asylum, of whom 276 were males
and 224 females.   With very few exceptions, I have the
weights of the heart in all these cases, and we shall first see
what indications may be observed from an examination of
them.

Weight of the Heart.—It is quite easy to imagine that an
amount of organic heart mischief, sufficient to cause distur-
bance of the cerebral and general circulation, and to give rise
to symptoms which may react on the brain and cause or
predispose to insanity, may not only exist during life undis-
covered amongst the insane, but may elude a perhaps not
over-careful examination of the valves and openings between
the cavities of the heart, after death.   Under these circum-
stances, then, and just as, during life, hypertrophy of the
heart, with murmur, is generally considered to be one of the
surest signs of organic disease of its valves, so, after death, its
weight affords special indications of great value.   In all these
cases due allowance must, of course, be made for the weight
and age of the patient, the state of his blood, and the
presence of kidney disease.   I cannot go so minutely into the

subject as to take the general body weight into consideration
for comparative purposes. Sufficient indication as to this
will be afforded by the ages of the cases, according to which
I shall classify and average the weights of the heart.
The existence of such kidney disease—viz. chronic Bright's
disease with albuminuria and a certain amount of uræmia—
as may give rise to hypertrophy of the heart, is extremely
rare amongst the insane, and of very little consequence as
a disturbing element in a consideration of the causes of
hypertrophy of the heart amongst them. I only know
of three instances, out of over 1,400 patients in the
West Riding Asylum at present, in which there is chronic
Bright's disease with albuminuria; and, out of 102 cases of
epilepsy, in which the urine was carefully examined by
Dr. Aldridge, about two years ago,[1] in only two was there
albumen present, and then only *traces* of it, and, so far as
can be ascertained, unconnected with any permanent kidney
disease. Out of the whole 500 cases, moreover, whose *post-
mortem* records I have searched, I can only find 7 in which
Bright's disease or uræmia was certified as a cause of death,
and, in two of these cases, the disease was puerperal and
acute. Hypertrophy of the heart among the insane, then,
when not associated with valvular disease, may be referred, as a
rule, to the reactions and strain entailed upon the organ by the
existence of mental insanity, arising as it does from varying
diseases of the brain, in some of which the systemic circula-
tion is interfered with, whilst in others there is more or less
rapid wasting of the brain substance, and loading of the
blood with effete materials—the outcome of this wasting, as
well as of that of the tissues generally, when rapid emacia-
tion sets in. As to these reactions of the brain on the heart,
and *vice versâ*, of the heart on the brain, I shall have to speak
further on, so need not now digress. I shall acquaint the
reader, in the simplest and shortest way, with my results, as
to the weight of the heart amongst the insane, by embodying
them in a Table (No. V.), in which I have calculated the
average weights of the heart at different ages, and placed
them, for comparative purposes, side by side with those of

[1] 'West Riding Asylum Reports,' vol. i. 1871, p. 100.

TABLE V.

Showing the Average Weight, in Ounces, of the Heart at different Ages; the Numbers, on which the Averages are calculated, being placed in adjacent Columns.

Age	Marylebone Infirmary and Somerset Asylum Males		Females		Somerset Asylum only Males		Females		West Riding Asylum Males		Females		Mania Males		Females		Melancholia Males		Females		Epilepsy Males		Females		General Paralysis Males		Females		Disorganisation of Brain[1] Males		Females	
Years 15 to 20	7·61	18	8·46	15	··	··	··	··	8·94	8	7·50	3	··	··	··	··	··	··	··	··	··	··	··	··	··	··	··	··	··	··	··	··
20 ,, 30	10·06	58	9·08	74	8·75	46	7·57	29	10·67	20	7·87	24	9·5	4	8·17	6	··	··	10·12	4	10·83	9	7·37	8	··	··	··	··	··	··	··	··
30 ,, 40	11·36	118	9·45	87	9·94	59	7·73	49	10·96	63	9·17	53	10·87	8	9·76	18	9·0	2	8·94	8	11·0	11	8·88	9	10·95	28	9·42	6	11·30	10	8·37	4
40 ,, 50	11·53	137	9·6	106	10·75	76	8·31	49	11·56	75	9·46	46	10·67	12	9·42	13	12·50	7	9·19	8	11·12	4	9·28	8	11·78	40	10·58	6	11·87	12	9·87	8
50 ,, 60	11·83	119	10·44	106	11·22	42	9·15	39	12·71	48	10·43	31	12·22	9	9·95	10	10·93	7	10·43	7	··	··	··	··	13·27	12	··	··	12·59	17	11·25	10
60 ,, 70	12·94	126	10·64	149	12·42	39	9·12	41	14·14	42	10·22	34	16·54	12	9·82	11	13·12	8	10·91	6	··	··	··	··	··	··	··	··	13·23	23	10·58	12
70 ,, 80	13·14	100	10·10	150	11·52	21	9·36	20	14·70	12	10·82	22	16·0	2	11·25	7	··	··	··	··	··	··	··	··	··	··	··	··	13·35	7	11·07	13
Above 80	12·1	24	10·27	76	13·35	7	9·46	5	13·62	2	13·87	4	·	··	··	··	··	··	··	··	··	··	··	··	··	··	··	··	··	··	14·0	3
Totals and s)	11·83	700	9·86	763	11·14	290	8·67	232	12·62	270	10·26	217	12·56	47	9·73	65	11·39	24	9·92	33	10·98	24	8·51	25	12·0	80	10·0	12	12·47	69	10·85	50

Average weight of healthy adult human heart { REID, male, 11 ozs. female, 9 ozs.
{ PEACOCK. ,, 9½ ,, ,, 9 ,,

According to CLENDINNING, the healthy human heart continues to increase in weight up to an advanced period of life; but at a comparatively slower rate subsequently to the age of 29 years.

[1] Deaths of consecutive dements with wasting, and of chronic dements with chronic atrophy, of the brain.

Dr. Robert Boyd,[1] founded on 2,614 *post-mortem* examinations at the Marylebone Infirmary and the Somerset County Lunatic Asylum.

I have, further, classified the weights in my own—West Riding Asylum—cases, under different headings in the Table, according to the nature of the disease, still arranging them according to their ages. The careful calculation and compilation of this Table has cost me no little trouble, and I shall now proceed to draw attention to the leading facts indicated by it. Taking the highest average standard weight of the adult heart to be, according to Reid, 11 ozs. in the male, and 9 ozs. in the female, it will be observed that the *mean* average weight of that organ (in the calculations for which I have always excluded the cases below 20 years of age) was, in 1,463 cases dying in the Marylebone Infirmary and Somerset County Asylum (about one-fifth of whom were insane) for the males 11·83 ozs., and for the females, 9·86 ozs. ; on 522 insane persons dying in the Somerset Asylum, it was somewhat less, being, for the males, 11·14 ozs., and for the females, 8·67 ozs. : whilst, in 487 insane persons dying in the West Riding Asylum, it was considerably more than at either of the other places, being as much as 12·62 ozs. for the males, and 10·26 ozs. for the females. We see, then, that, excepting in the female insane of the Somerset Asylum, the mean average weight of the heart was in all cases greater than Reid's (the highest) average healthy standard, being in the West Riding Asylum insane not only very considerably greater (nearly 1 oz. for each sex) than amongst the mentally sane, but even more so when compared with that of the Somerset Asylum insane. The numbers on which the mean averages are calculated are much the same for the West Riding as the Somerset insane, and I am somewhat at a loss, therefore, to account for this excess of average weight of the heart amongst the West Riding insane, especially as I find that, according to Haviland, heart disease is more common in the county of Somerset than in the West Riding

[1] 'Tables of the Weight of the Human Body and Internal Organs,' &c., 'Philosophical Transactions,' 1861, p. 241. I am indebted to Dr. Boyd for a reprint of this valuable communication to the Royal Society.

of Yorkshire. The fact, however, remains, be the expla-
nation what it may. As we shall shortly see, the mean
average weight of the heart is greater in some forms of in-
sanity than others, and it might possibly be due to a greater
prevalence of the former, or less frequency of the latter, in
Yorkshire, as compared with Somerset. The mean average
weight of the heart, for both sexes, and for all ages above
twenty years, in different forms of insanity, is as follows :—
1st, Disorganisation of Brain, 11·66 ozs. ; 2nd, Mania, 11·14
ozs. ; 3rd, General Paralysis, 11·0 ozs. ; 4th, Melancholia,
10·65 ozs. ; and 5th, Epilepsy, 9·74 ozs. As the heart,
especially the heart of the insane, continues to increase in
weight up to an advanced period of life, it is evident that
the non-existence of epileptic cases above the age of fifty
will, to a certain extent, account for the comparatively small
average weight in their cases, whilst the common occur-
rence of disorganisation of the brain at a later age will so
far explain the increased average weight of the heart in
cases of that disease. But, to look at the matter from
another aspect, let us now take only those ages—viz. thirty
to fifty years—during which period all forms of insanity are
most active and prevalent, and epilepsy and general paralysis
have their greatest, and for the most part, only prevalence ;
and we shall get somewhat different results. General para-
lysis being an easy first,[1] with a mean average weight of the
heart, for both sexes, of 10·68 ozs. ; disorganisation of the
brain, second, with 10·35 ozs.; mania, third, with 10·18 ozs.;
epilepsy, fourth, with 10·7 ozs.; and last, melancholia, with
9·90 ozs. These results would seem to indicate a condition
of hypertrophy of the heart amongst general paralytics, as
well as amongst patients the subjects of chronic disorganisa-
tion, wasting, or atrophy of the brain, in many of which
cases there exists multiform disease of the arterioles and
capillaries of the brain, and obstruction to the systemic
circulation, not to speak at present of the effects of past or
present mental and physical excitement in causing strain of

[1] This is all the more striking in general paralysis—a wasting disease in its
latter stages—where we should expect to get, as in phthisis, a certain amount of
concurrent atrophy of the heart.

the heart, and of the loading of the blood with that effete material which, sooner or later, occurs from the waste of the brain and of the tissues generally.

Let us pass on, now, to see to what extent this excessive mean average weight of the heart, amongst the insane, can be accounted for by organic disease, other than that of the brain itself, and to further analyse my 500 West Riding Asylum cases, so far as the condition of the heart after death is concerned. In no less than 241 cases out of the 500, or nearly half, the heart was above the average healthy standard weight of 11 and 9 ozs. respectively. This could be more or less accounted for, as follows, by

	Males	Females	Total
Valvular disease of heart	72	33	105
Aortic disease	22	9	31
Excess of external fat	5	9	14
Hydro-pericardium or adherent pericardium . .	4	6	10
Co-existing disease of the kidneys or lungs . .	4	5	9
No heart, aortic, or other than the cerebral disease	42	30	72
Total	149	92	241

Of these, 72 cases (nearly one-third of the 241), in which there was no disease that might account for the hypertrophy (in such cases as there was a real hypertrophy) except the cerebral disease, in so far as this could react on the heart directly or indirectly, the nature of the insanity was as follows :—

	Males	Females	Total
General Paralysis	13	4	17
Disorganisation of brain . . .	8	8	16
Epilepsy	10	3	13
Mania	6	7	13
Melancholia	4	8	12
Idiocy	1	0	1
Total . . .	42	30	72

It will be observed that, amongst the males, general para-

lysis had the greatest number, and that no less than one-half laboured under general paralysis and disorganisation of the brain ; which tends to show that the excessive weight of the heart, as I have ascertained it to exist in these two diseases, cannot be entirely attributed to organic disease of the valves or openings of the heart, or to advanced age, and must, to a certain extent, be referred to the peculiar conditions of the brain found to exist in those cases, and to other conditions, concerning which we shall have more to say further on. Even amongst the females, where general paralysis is very rare, we find that considerably more than one-third suffered from these two forms of insanity, just about one-fourth of the 30 being cases of disorganisation of the brain. The fact also that epilepsy, a rare disease after fifty years of age, ranks next highest in this category, adds further strength to the supposition that this hypertrophy is not, in the main, the natural condition of advanced years.

Besides the 136 cases, in which the excess over the average weight of the heart was associated with valvular or aortic disease, there were also 44 cases (24 males and 20 females) in which, though the heart was not above the average weight, yet there was similar disease of the valves or aorta. I may state that I include under this term—disease of the valves or aorta—constrictions and dilatations of the valvular openings and aorta, as well as any decided roughening or calcareous deposits, with atheromatous or fibroid patching and thickening, though not, perhaps, amounting to constriction or dilatation. We thus get a total of 180 cases out of the 500, or more than one-third, in which there was some organic disease of the cardiac valves (mostly of the left side) or of the aorta, observed after death. As to the nature of the hypertrophy, where it existed, whether it was concentric or eccentric, unilateral or general or composite, the recorded facts are, in many instances, too vague to afford precise information. I gather, however, as a general result, that concentric hypertrophy of the left ventricle was most common, and that eccentric hypertrophy of the right ventricle was very common ; whilst, in a good many instances, these conditions seemed to be combined. Other forms, or combinations, of

nypertrophy were rare. Recent pericarditis was very rare, but *post-mortem* evidences of the former existence of pericarditis were very common; there being from 2 to 4 fluid ozs. of serum in the pericardial sac in 22 cases; above 4 fluid ozs. in 10 cases; fibrous thickenings of the visceral layer of the pericardium in 39 cases; adherent pericardium in 21 cases; and a coating of shaggy lymph round the heart in 4 cases. In several of the above cases, one or more of the conditions described were combined in one case, but, for convenience, I have stated now one and now the other condition, so as to give a tolerably fair idea of the state of matters. The substance of the heart is variously described, in many cases, as being pale, soft, or flabby, and, in many others, the walls are described as being thin; but, as microscopic examination was very rarely resorted to, it would be very difficult to state with any amount of accuracy in how many instances the substance was really fatty.[1]

I pass on now to consider—

HEART DISEASE AS IT EXISTS AMONGST THE LIVING INSANE.

My observations, under this head, are founded on an examination into the state of the circulation generally, and condition of the heart, in a little more than 1,100 cases of insanity in the West Riding Asylum. Of this number the majority, 680, were men. In order to free my results from as much fallacy as possible, and to obtain uniformity, I took care to exclude all patients suffering from acute or severe bodily disease, and those taking medicines which would

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[1] With regard to the state of the heart and frequency of heart disease, as observed after death, amongst the insane elsewhere, or the sane generally, recorded facts are meagro, vague, and unavailing to me for purposes of comparison and contrast. According to Dr. Sutherland, in his Croonian Lectures published in the 'Journal of Mental Science' for July 1861, the total number of *post-mortem* examinations, from March 1853 to August 1856, at St. Luke's Hospital, was forty-two, and disease of the heart was found in thirty-four, so that in only eight cases did the heart present a healthy appearance. According to M. Foville, five out of every six, and, according to Romberg, five out of every seven hearts, of cases of insanity, which these observers had examined, were found to be diseased. The results of my examination of the West Riding Asylum records would seem to indicate quite as large a proportion of diseased hearts amongst the insane.

materially affect the condition of the pulse or action of the heart. I was also careful, in all cases, to examine the pulse and the heart while the patient was in the sitting posture, and all my cases were examined between one hour after dinner, and one hour before tea—viz. between 2 and 5 P.M. My method of procedure was as follows, with regard to the men :—Being first stripped of jacket and waistcoat, and their braces turned back, they were placed in sitting posture in a chair : I then observed the condition of the general circulation as evidenced by the state of the hands and face, and noted this, with the name and other particulars, in a book before me. By the time that I had done this, they had, as a rule, regained any composure which they might have lost, through the exertion of divesting themselves of their garments, and a little natural surprise at what was going to be done to them, and I then examined the pulse ; after which, the shirt was pulled well up, in front, and a careful examination of the heart made, as to the nature of its sounds, the situation of the apex beat, and the impulse as communicated to the hand or seen. The women, who were all also examined in the sitting posture, had to be dealt with in a more delicate, and, of course, less satisfactory manner, the frock, &c., being sufficiently opened in front to enable one to listen at the apex and base of the heart. The women were also very much more apt than the men to become flurried during the examination, and my results, as to the condition of the pulse in them, are also less satisfactory than amongst the men. I am afraid, therefore, that I must generalise principally from the results of my examination of the males alone, except in so far as I can use the results amongst the females with tolerable freedom from fallacy ; but as I only examined 450 women as compared with 680 men, this does not give me much concern, and I think it will be admitted that the basis is sufficiently large, amongst the men alone, for the purpose of a fair generalisation. With regard, then, to the

Pulse and State of the Circulation generally,

My results tend to ratify a preconceived idea of my own, which I had held on *primâ facie* grounds—viz. that chronic insanity is essentially of an *asthenic* nature ; or rather, to express myself more correctly, I should say that, in a great majority of the cases of chronic and advanced insanity, especially where it is accompanied by consecutive dementia, it appears to me there is a certain amount of asthenia. The weakness and impairment of the mental faculties, and of the functions of the nervous system generally, are almost always associated with a corresponding diminution of vital power and enfeeblement of bodily function. Putting aside, for the present, cases of acute dementia and atonic melancholia, in which we get the same condition more intensified, but at the same time of a more temporary nature, I may state that, in the subjects of advanced general paralysis, chronic epilepsy, of consecutive dementia, and of advanced chronic insanity generally—constituting about three-fourths of the bulk of an asylum population—we almost invariably get evidences of languor of the circulation, as shown by more or less coldness and lividity of the hands, often of the face, and I have no doubt very frequently of the lower extremities. This condition, of course, varies in intensity according to the nature of the case, and stage to which it has advanced, and is, no doubt, more or less affected by the state of the heart. Though I believe it to be primarily and essentially of neurotic or cerebral origin, yet there can be no doubt the condition is often aggravated by the habits of the patients themselves, many of whom, from this very languor, are indisposed to exercise, and prefer standing moping against a wall or in a corner, with the arms pendent by the sides, or sit quietly, staring into vacancy, with their hands spread out before them on their knees. This condition, as well as that of the pulse, to which I shall shortly refer, seems to be regulated more by the stage and nature of the insanity, than by the bodily health and condition of the patient. In the majority of these cases, too (excluding those with heart disease or with corded arteries), I have been obliged to describe the

pulse as feeble and small, or, when full, compressible. The condition, with regard to this, likewise varied in intensity according to the stage and duration of the insanity, being, of course, most marked in advanced demeuts. In illustration of this purely cerebral or neurotic depression of the circulation, as it exists amongst the insane, I shall venture to give the details of a full examination of two male patients, in which it was present to a maximum degree of intensity. They were the only two cases in which I could not feel the ordinary radial pulse at all, though in many it was *just* perceptible, and able to be counted. Both of the men were stout and well-nourished.

Case I.—W. M—, No. in Register, 5295 : æt. 50, admitted April 14, 1868 ; being melancholic, and having delusions of a suspicious nature. and symptoms of locomotor ataxy. Since that time, the ataxic symptoms have gradually intensified, so that for the last two years he has been unable to walk at all, and has had no control over the bladder. During this time he has become gradually more demented, and is now less suspicious and more cheerful than he was. He sits all day, contentedly, in a wheeled chair drawn up by a table, to and from which he is wheeled in the chair. By means of an india-rubber urinal he is kept dry during the day, and he is wheeled to the closet always last thing before going to bed. I must add, that since October 5, 1870 (according to the note-book), he has been almost totally blind ; but as to how his sight was before that time, there is no record to show. He is very ' hearty ' as to his appetite, and, apparently, well-nourished and in good bodily health. He is not pale or anæmic-looking, but there is some slight chilliness, with lividity of the hands. There are all the symptoms, then, of a profound and general affection of the nervous system, without any cardiac (as we shall see) or other bodily disease. The condition of his circulation and state of his heart were as follows :—As he sits in his chair, a most careful and prolonged examination fails to lead to the perception of a radial or ulnar pulse on either side, neither also can the brachial pulses be felt ; the femoral and posterior tibial pulses, on both sides, are also imperceptible. The temporal pulse is imperceptible on the right side, and is *just* to be felt on the left side, being 104 per minute, and thready in character ; the apex beat of the heart is nowhere distinct, and the action of the heart is, so far as can be made out, somewhat tumultuous ; the second sound of the heart is best heard, and both sounds are heard equally well to the right as to the left of the sternum, though there is no other evidence of hypertrophy or dilatation ; there is no murmur to be heard, nor are there any signs or symptoms of aneurism ; the corneæ are rather cloudy, but there is no *arcus senilis,* nor is there any atheromatous cording of the arteries to be felt anywhere.

Case II.—G. B—, No. in Register, 5872 : æt. 45, admitted October 29, 1870 ; in a state of dementia, with restlessness and incoherence, and being partly paralysed in the right side. The history brought with him was that,

in July 1869, he had had a paralytic stroke affecting the right side of the body, and that he had not been able to follow his employment since. For about six months before admission, he had been restless and talked almost incessantly and incoherently to himself. About a week before admission, he became quite unmanageable, having acquired strange fancies and become the subjects of hallucinations of hearing, and he was taken to the workhouse. Since admission he has gradually got more and more demented, and there has been very little excitement; at the present time he is very demented, never speaks, and sits most of the day, rather inclined to the left side, staring vacantly downwards. His face is utterly devoid of expression, and there is everywhere, except at the extremities, a deadly pallor of the surface of the body ; so that the face, were it not for the open eyes, might be taken for that of a dead person. He is well-nourished, and can readily be roused to take his meals (which he does with evidently fair appetite), and to dress and undress himself, and attend to the calls of nature. Though still weak on the right side, and occasionally troubled with choreic twitchings of the muscles of that side, he can manage to get about without assistance ; he suffers from no disease except that which is of cerebral origin, and he has been, for the last two years, in very much the same condition as above described. The condition of his circulation and state of his heart are as follows :—The hands and feet are cold, swollen, and mottled with patches of lividity ; the radial pulse, on the right side, is *just* perceptible, 84 per minute ; on the left side it is imperceptible. The ulnar pulse is imperceptible on both sides ; the brachial pulse is quite distinct on the right, but imperceptible on the left side ; the femoral pulse is perceptible on both sides, but the posterior tibial can, on neither side, be felt. The temporal pulse is not to be felt on either side. Both sounds of the heart are distinct and somewhat accentuated, especially at the base, but the apex beat cannot be defined : there is no murmur to be heard, nor are there any signs or symptoms of aneurism ; there is no *arcus senilis*, or cording of the arteries.

Such, then, is the condition of quiet vegetation (I might almost term it such) to which a man may be brought by means of advanced disease of the cerebrum and nervous system. As it is, undoubtedly, in both these cases of cerebral and neurotic origin, so, also, is it in the large number of the chronic insane, amongst whom it exists to a smaller extent ; and it is a condition which, unfortunately, is generally of too profound an origin to admit of radical treatment ; and, by its reaction on the cerebrum, it not only tends to maintain itself, but, at the same time, to intensify the brain mischief : and, thus, by the mutual re-action of the two conditions, the one on the other, we arrive at doubly pernicious results.[1] Further

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[1] It is to this condition that we must look for an explanation of the so-called cerebral fainting, from time to time observed amongst the insane. I am informed

evidence of this depression of the circulation and general
lowered vitality, common amongst the chronic and advanced
insane (whose natural tendency to, and fondness of, basking
in the sun is well known), might, I believe, be obtained from
carefully conducted thermometric observations.   I have cer-
tainly ascertained, from continued thermometric observations
in several cases of senile dementia, that the temperature is
lower than the average ; but here, no doubt, it is only one of
the symptoms of a natural decline, which is, as I think, often
induced, prematurely, by advanced cerebral disease.   I can-
not speak further with regard to this from experience ; and,
even if I could, it would not be within the scope of this paper
to do so; but I may add that, in both of the remarkable
cases, details of which I have above given, the temperature
of the axilla was considerably below the average, being in
Case I. (with a thermometer which registered my own tem-
perature as $98\frac{3}{5}°$ F.) $97\frac{2}{5}°$ F., and in Case II., $96\frac{2}{5}°$ F.

In 46 out of the total number of cases examined I find
that I have recorded the pulse to be *very* weak or just per-
ceptible. The form of disease in these cases was as follows :—
Epilepsy, last stage, 16 ; consecutive dementia, far advanced,
12 ; melancholia, 7 ; disorganisation of brain or chronic
atrophy, 2 ; general paralysis, 3rd stage, 3 ; senile dementia,
1 ; acute dementia, 1 ; idiocy, 1 ; and acute mania, 3.   In no
less than four of these cases—2 young, but far advanced
epileptics, 1 general paralytic, and 1 senile dement—the
right [1] radial pulse was imperceptible, though there was no
thoracic disease or physical abnormality to account for the
condition in any of the cases.

If I were asked to arrange the patients in classes accord-
ing to their priority as to this condition of the circulation, I
should do so as follows :—1st, advanced epileptics ; 2nd, in-
tense and passive melancholics ; 3rd, advanced consecutive

that Case II. is subject to attacks of this kind, though I have not myself seen him
in one.   I have, however, in two other cases (one chronic melancholic, and one
chronic dement) observed slight fainting fits to occur on several occasions ; and in
neither of these cases is there any evidence of organic cardiac or arterial disease,
though there is a permanent condition of great languor of the circulation.

[1] It is noteworthy, that when only *one* radial pulse could be felt, it was always
the *right* which was imperceptible.

dements ; and 4th, advanced general paralytics. In other forms of disease it is either rare or not, as a rule, to be properly estimated on account of disease of the heart or cording of the arteries. In a large proportion of the cases above 50 years of age there was cording of the arteries. If I exclude those cases in which the result was attributed by me to nervousness, I must state that I found a pulse of above 100 per minute to be of exceedingly rare occurrence ; just, of course, what we would naturally expect to find amongst persons free from acute bodily disease.

Temporary quickening of the pulse from nervousness, and some surprise or apprehension with regard to the examination, was principally observed amongst the women, with whom, especially the convalescent and less advanced cases, it was the rule rather than the exception ; it was also not uncommon amongst the sensible and less advanced male cases. With regard to the great majority, however, and especially in those cases in which the pulse was abnormally slow, the examination seemed not to make the slightest difference in the pulse, for, where the pulse was abnormally quick or slow, I always re-examined it after the temporary surprise and effects of the little exertion had passed away. This temporary increase in the frequency of the pulse was so marked, by its presence, in convalescent and fairly intelligent patients, and, by its absence, in the more chronic and advanced cases, that, in the process of time, I felt I could readily, blindfolded, have diagnosed the condition of the patient from an examination of the pulse alone. Considering, therefore, the many fallacies which beset the subject, I shall not go further into the condition, which certainly existed in many cases, of abnormal frequency of the pulse. With respect to abnormal *slowness* of the pulse—the condition which we shall next proceed to investigate—the case is, however, different, and the subject is extraordinarily free from fallacy, physical exertion, or mental emotion, having a tendency rather to lessen than to intensify the condition. It is as a rule purely of neurotic or cerebral origin, and is, as we are all aware, occasionally observed in healthy people, as a functional neurosis or natural idiosyncrasy. Let us, then, examine

into its occurrence amongst the insane ; and, taking the average frequency of the pulse to be 72 per minute, we shall ascertain in how many and what sort of cases the pulse was below 70 per minute, premising, however, that this condition of lowered rhythmical action of the heart must be considered quite apart from diminished energy in its propelling powers, the two conditions being quite distinct, and by no means invariably associated with one another. The pulse was below 70, in 184 cases out of the 1,100 examined, or in just a little more than one-sixth ; being, in 117 cases, below 70 and above 60, and, in 67 cases, 60 or below. The lowest number of beats per minute observed was 52, and this rate of the pulse I have recorded in 7 cases. Abnormal slowness of the pulse was decidedly proportionately most frequent amongst melancholics, imbeciles, and advanced epileptics, amongst the latter of whom I include epileptic idiots ; and, so far as I can judge from my observations, it was more common amongst the men than the women. Absolutely, of course, the greatest number of cases of it occurred amongst the consecutive dements and amongst persons of middle age. It was only found to exist in two instances amongst general paralytics, in which cases the pulse was respectively 52 and 56 per minute. It was the prevailing condition of the pulse amongst quiet imbeciles and sullen and morose men. Other changes in the pulse, such as irregularity in volume, inter-mittency, jerkiness, &c. were very rare, when not associated with heart disease ; but simple irregularity, as to frequency, however, was not at all uncommon, being most frequently observed in melancholia.

Let us pass now from a consideration of the state of the heart, as evidenced by the distal circulation, to that of the condition of the great central organ itself, as ascertained by an examination of the chest.[1] I shall first dispose of the 450 women I examined, in whose cases I gained very little further information than the application of the stethoscope in the region of the apex and at the base of the heart could afford me. Distinct replacing murmur was heard to exist in 28 cases ; and, excepting one case in which the

[1] For obvious reasons, amongst the insane, I have confined my enquiries to the objective symptoms alone.

murmur was clearly hæmic and due to spanæmia, and another in which it was double and heard loudest at the base, the murmur was single and apparently due to organic disease of the heart, being apicial systolic in 13 cases, apicial diastolic in 1 case, basal systolic in 10 cases, and basal diastolic in 2 cases. Organic disease, sufficient to produce distinct replacing murmur, and associated with more or less hypertrophy of the heart (as evidenced by the situation of the apex, impulse of the heart, and nature of its sounds), existed, then, in just 6 per cent. of the females examined. To this number must be added 63 cases, or just 14 per cent. more, in which the heart was damaged to a less extent, and in which hypertrophy less frequently existed. In these latter cases the sounds were modified, rather than replaced, by a murmur, and I have variously described them as being blowing, rasping, grating, rough or broken in character. This slighter condition was double and basic in 2 cases, and, in the rest, single ; being apicial systolic in 35 cases, apicial diastolic in 1 case, basal systolic in 14 cases, and basal diastolic in 11 cases. We thus get a total of 20 per cent., amongst the women, in which there were physical signs of more or less heart mischief. *Du reste*, so far as the females are concerned, more or less hypertrophy was indicated in 37 cases, or a little more than 8 per cent., by the nature of the sounds and impulse of the heart, independent of organic murmur or modification of the sounds. In 16 more cases the sounds of the heart are described as being muffled and indistinct. Accentuation of the sounds (whatever that may signify) was not at all uncommon amongst the females, both at apex and base ; but, as this is a comparative condition, and requires more of the *auditus eruditus* for its correct appreciation than I pretend to possess, I shall not go into further detail, either here or with regard to the men, as to my observations on this point.

I am sorry that, for various reasons, I am obliged to dismiss the women in this general manner, and pass now to examine into the conditions of the heart amongst the 680 men whom I was enabled to examine much more fully and satisfactorily, and with whom I am much better acquainted, generally, than with the women. Out of the 680 cases, I

found distinct replacing murmur to exist in 53, or just about 8 per cent. of the total number ; whilst in 103 cases, or 15 per cent. more, what I have called a modifying murmur, or abnormal alteration of the sound, was present, making altogether 23 per cent. of the total cases in which there were physical signs of more or less valvular disease of the heart. The situation and time of the murmurs in these two classes of cases were as follows :—

	Replacing Murmur	Modifying Murmur	Total
Apicial systolic	27	82	109
,, diastolic	2	...	2
Basal systolic	16	10	26
,, diastolic	7	8	15
Apicial double	...	1	1
Basal ,, ...	1	1	2
Combined apicial systolic and basal diastolic	...	1	1
Total	53	103	156

With regard to the cases in which the murmur was replacing, there were only 6 in which there were no physical signs of hypertrophy of the heart, a condition which existed to a greater or less extent in all the other cases, as shown by displacement downwards of the apex, increase in the impulse, and heaving, of the heart, with intensification of the sounds. In the majority of cases, the displacement of the apex was downwards and inwards, the apex beat being at or near the epigastrium in 12 cases. In only 7 of the cases was the apex in the nipple line or *outside* of it. With regard to other objective symptoms, I may state that 2 of these cases have had fainting fits, and that a third has had general dropsy, but is now up and about again, having improved under the influence of digitalis. Passing to those cases in which the murmur was modifying and not distinctly replacing, I may add that there were signs and symptoms of more or less hypertrophy in the great majority of these cases ; for, as a rule, I did not pay much attention to modifications of the sounds unless they were accompanied by some hypertrophy of the heart, as ascertained by the position of the apex, and

nature of its impulse. And here again the displacement of the apex was much oftener downwards and *inwards* than downwards and outwards, being, in no less than 29 cases, at or close to the epigastrium, in 11 cases between the 7th and 8th ribs, midway between the epigastrium and nipple line, and in only 6 cases in or just outside the nipple line. Premising that there was no evidence to show that the murmur was hæmic in any of the male cases, I shall next show (in Table VI.) the percentage of cases of each disease (in which a sufficient number was examined for generalising purposes) in which the murmurs occurred.

TABLE VI.

Form of Disease	Number of Cases examined	Replacing Murmur heard in, per cent.	Modifying Murmur heard in, per cent.	Total
Melancholia . .	56	16·07	32·14	48·21
General paralysis .	43	13·95	18·60	32·55
Recurrent mania .	72	9·72	18·05	27·77
Epilepsy . . .	99	6·06	12·12	18·18
Dementia . . .	198	6·56	10·10	16·16
Chronic mania . .	95	4·21	10·52	14·73

It will be observed how decidedly more prevalent heart disease was in *melancholia* than in any other form of mental or cerebral disease; no less than one-sixth of the melancholic cases having distinct replacing murmur, and two-sixths more having a modifying murmur; making, in all, nearly one-half in which the heart was more or less affected. Hypertrophy with murmur was more common and more extensive in melancholia [1] than any other disease, the murmur generally being apical systolic, and the apex displaced, downwards and inwards, being at or close to the epigastrium in no less than 3 cases out of the 9 in which the murmur was replacing, and

[1] This scarcely is in accord with my results as to the weight of the heart after death in the different forms of insanity; but, as we well know, the tendency for melancholics is, if they do not quickly recover, to lapse into a condition of consecutive dementia, and become more or less anæsthetic as to subjective symptoms. So that these melancholics would be classed at the time of death amongst the cases of disorganisation of the brain, in which, as we have seen, the weight of the heart after death is considerably above the mean average. This would, I think, explain the discrepancy.

in 10 out of the 18 cases in which it was modifying; and betwixt the 7th and 8th ribs in 2 of the former and 3 of the latter. I shall have more to say, further on, about this displacement, downwards and inwards, towards the epigastrium, of the apex of the heart; for it was by no means uncommon amongst other patients in whom there was no murmur to be heard or evidence of lung disease. With regard to the other forms of insanity, I may just add that, in General Paralysis, the replacing murmur was basal *diastolic* in the majority of the cases (4 out of 6), whilst, with rare exceptions, in the other forms of disease, it was either apicial systolic or, less frequently, basal systolic. None of the 15 senile dements examined had replacing murmur; but no less than 8 of them had modifying murmur with more or less hypertrophy of the heart, the murmur being apicial systolic in 5 cases, and basic *diastolic* in 3 cases. In other less frequent forms of mental disease, not mentioned in Table VI. and not already referred to, I may, perhaps, show, at a glance, the results, as follows :—

Disease	Cases examined	Replacing Murmur in	Modifying Murmur in
Recurrent melancholia	4	1	2
'Folie circulaire'	8	1	1
Imbecility	23	2	0
Acute dementia	3	1	0
Dementia, with present or recurrent excitement	42	0	8
Acute mania	4	0	3
Total	84	5	14

Here again, as will be seen, where melancholia comes in, there is a great proportion of heart disease. I may further state that, out of 12 convalescent patients examined, 2 had distinct replacing murmur—in both cases apicial systolic.

Having now examined the cases in which there was organic murmur, with more or less hypertrophy, I shall proceed next to notice the occurrence of other abnormalities of the heart, as ascertained on examination. In 72 of the cases in which there was no murmur (excluding the very stout men,

in whom, naturally, the sounds are with difficulty heard), or in $10\frac{1}{2}$ per cent. of the whole number, I have had to describe the heart's sounds as muffled, very feeble, or indistinct, and the apex beat as very feeble or ill-defined: these cases consisted for the most part of chronic dements, far-advanced epileptics, and melancholics. This condition was found in no less than one-seventh of the melancholic patients.

In no less than 144 of the 680 patients examined— or about 21 per cent.—there was more or less hypertrophy of the heart present without the existence of murmur to account for it, as shown by the position of the apex, nature of its impulse, and character of the sounds. It will scarcely be credited, when I state that in such a large proportion as 63 of these cases, the apex beat was under, below, or very close to, the ensiform cartilage, in the epigastric region, with thumping impulse and loud coarse sounds. In 9 of the rest it was between the 7th and 8th ribs further outwards, and, in the remainder, it was below the 6th rib, generally about half way between the epigastrium and the left nipple line, being, in only a few instances, in, or outside of, the nipple line. Proportionately to their numbers, epilepsy, general paralysis, and melancholia were largely represented in this category, but in these diseases, this hypertrophy, of uncertain origin, existed in its milder forms. It was in chronic dements, chronic and recurrent maniacs of middle or old age, that this form of hypertrophy was absolutely most common and most extensive. On looking carefully over my list, I can only find about six or seven cases in which this hypertrophy might be due to chronic disease of the lungs; and, excluding these, I shall now analyse the cases in which the apex beat of the heart was at, or close to, the ensiform cartilage, taking only, for present purposes, those cases in which the hypertrophy was not presumably due to heart disease; and although organic murmurs are not always permanent, and may have existed in some of these cases, indicating a sufficient amount of valvular disease to give rise to the hypertrophy without my having heard them when I made the examination, yet I must confine myself to the ascertained facts. Of these 57

cases, then, about half were between the ages of 30 and
50, about one-third were 50 years of age or above, and
about one-fifth were below 20 years of age.   No less
than 46 of the cases were consecutive dements, or chronic
and recurrent maniacs of old standing.   In only 6 of the
cases was the residence in the asylum under one year, and
3 of these were transfers from asylums where the patients
had resided some time; 27 had resided above five years, and
the rest between one year and five years.   Thus the great

TABLE VII.

Form of Mental or Cerebral Disease	With Replacing Murmur	With Modifying Murmur	No Murmur present	Total
Dementia, consecutive, and with present or recurrent excitement	5	6	24	35
Chronic and recurrent mania .	3	12	25	40
Melancholia	3	6	2	11
General paralysis . . .	1	1	2	4
Epilepsy	...	2	2	4
Senile dementia	...	2	...	2
Idiocy	...	...	1	1
' Folie circulaire ' . . .	...	...	1	1
Total number of cases in which apex beat was in epigastric region	12	29	57	98

majority of the cases were chronic cases of old standing, in
which there was present excitement, or had been excitement
at some former time.   Table VII. shows the comparative dis-
tribution of cases in which the apex beat was close to, under,
or beneath the ensiform cartilage, in the epigastric region,
according to the nature of the cerebral disease, and the
presence or absence of cardiac murmur.   It shows how very
common, amongst the insane, hypertrophy or dilatation of
the right side of the heart must be, when no less than 98
cases altogether, out of the 680 men examined, or more than
14 per cent., presented this indication of its presence in a
very marked degree ; to say nothing of those cases—a large
number—in which the same condition was present, though
to a less extent.   The table also shows that this condition
was least common where there was replacing murmur, more

common where there was modifying murmur, and by far the
most common of all in those cases in which no murmur
existed ; and this is just what one would expect, for, in the
former cases, more or less hypertrophy of the left ventricle
would be present, which would tend to evert the apex beat,
whereas, in the latter, the much less frequent enlargement
of the left side would leave the apex beat free to be displaced
inwards to its full bent by hypertrophy or dilatation of the
right ventricle.   This condition of the right side of the heart
being common, unaccompanied by murmur and hypertrophy
of the left side, we should naturally expect it to be not
unfrequent as an *additional* affection of the heart in cases of
organic disease of the left side.   These combined affections
would so modify the apex beat as to bring it oftener down-
wards and inwards than downwards and outwards, and thus
explain the greater prevalence of the former than the latter
kind of displacement amongst the cases in which there was
replacing or modifying murmur, to which I have already had
occasion to refer ; for, had the disease of the left side been
uncomplicated, generally, we should have expected the apex
beat to have been more commonly displaced downwards and
outwards.

These results, as to the state of the heart amongst the
living insane, are corroborated by, and in striking harmony
with, those arrived at from my examination of the post-
mortem records.   It will also be observed, from a consi-
deration of Table VII., that in no less than 75 out of the 98
cases, this abnormal situation of the apex, with increased
impulse and intensity of the sounds, was found to exist in
consecutive dements (the majority of whom had at one time
laboured under more or less continued mental and physical
excitement), or were subject to recurrent attacks of such
excitement, and in the subjects of chronic and recurrent
mania.   We see, then, that there exists, amongst the
insane, a very large proportion of cases in which there is
more or less hypertrophy or dilatation of the *right* side of
the heart (not to be accounted for by valvular disease of the
heart, or lung disease) either alone, as it commonly is, or,
less frequently, associated with hypertrophy of the left ven-

tricle due to valvular disease and other causes. I say that it is unaccounted for by valvular disease of the heart, or lung disease, because we know that valvular disease of the right side is very rare and not, so far as my observations go, more common amongst the insane than the sane, and because I have made a very liberal allowance, in my calculations, for patients in whom there might be some obstruction to, or interference with, the pulmonary circulation. To what, then, are we to look as the cause of this peculiarly common condition of hypertrophy or dilatation of the right side of the heart amongst the insane ? Dr. Allbutt[1] has written on the subject of the effects of overwork and strain of the heart, and there was lately a good discussion on that topic after a paper on the subject read by Dr. Allbutt before the Clinical Society of London. It is, moreover, a subject which has altogether of late occupied considerable and increasing attention. Most observers are, I think, agreed that even spasmodic or recurrent *physical* excitement alone is a common cause of hypertrophy and dilatation of the right side of the heart amongst athletes and active workmen, whilst *continued* physical excitement and exertion are considered to be more productive still of this form of disease. Further researches into the etiology of this kind of heart disease, will, I believe, confirm the opinion of Dr. Farquharson[2] (who, at Rugby, has had abundant opportunity of observing the effects of both mental and physical excitement on the heart) that *mental* agitation, overwork, and excitement are more important elements in its causation than has hitherto been supposed. How much overwork and excitement of this kind, both mental and physical, there exists amongst the insane, is well known to all alienists, and how much more severe, prolonged, and continued it is amongst them than amongst the sane, is equally well known. Is it surprising, then, that the strain on the heart, which must inevitably result from this excitement and over exertion, should very often eventuate in more or less permanent hypertrophy or dilatation of the right side of the organ, amongst the insane ? I think not, and I am

[1] 'On Overwork and Strain of the Heart and Great Vessels.' Macmillan & Co., 1871.   [2] 'Lancet,' March 15, 1873.

at a loss to explain my results in any other manner. It is possible that, when years of more or less continued mental and physical excitement and over-exertion have passed over and the patient has lapsed into a state of quiet consecutive dementia, that there is a tendency for this condition of the heart to disappear ; for, as will be seen on referring to Table VII., considerably the largest number of those cases in which the condition was extreme, comprised the active maniacs, chronic and recurrent, although the consecutive dements were proportionately more numerous. It is to the existence of this condition of right-sided hypertrophy of the heart, as well as that of the left side which may be caused by advanced cerebral disease in its various forms, that I furthermore attribute the excessive mean average weight of the heart of the insane as compared with that of the sane. It would appear, then, that over and above, or mixed up with, the ordinary hypertrophy due to valvular disease of the heart, which is about the same amongst the insane as the sane, there are two forms of heart disease, associated with insanity, not attributable to any known cause except it be the cerebral disease, directly or indirectly. They consist in concentric or eccentric hypertrophy of the right and left ventricle respectively, the former being due indirectly to the cerebral disease, by its giving rise to over-exertion and excitement, and consequent strain on the heart, and the latter directly due to such obstruction to the systemic circulation as may be caused by the multiform and obscure diseases of the arterioles and capillaries of the brain, and other abnormal vascular conditions, as they exist in general paralysis, disorganisation of the brain, and advanced cerebral disease generally.

The amount of space which I have, rather unexpectedly, in my desire to look at the subject from all points of view, taken up in thus recording and analysing my results as to the state of the heart amongst the living insane, and in drawing attention to a few of their special indications, precludes my adding (as I had intended to have done in this paper) some further special remarks on heart disease, as to *how* it may act as a cause or modifying agent in cases of

insanity, and going into the subject of heart disease as a *result* of insanity, further or more fully than I have just done. With regard to the exact position which ought to be assigned to heart disease in the etiology of insanity, a full elucidation of this point could only be obtained by a careful examination of the symptoms on admission, and observing the nature of the insanity, in a considerable number of cases, of both sexes, in which the heart was diseased *at the time of admission.* I purpose examining 100 cases of each sex in this manner, and supplementing my present observations and remarks by a future paper on the subject, which I am convinced is of great importance and full of interest. Apart from this, however, it may be gathered from the results and facts which I have already recorded, that *melancholia* is the most common form of mental disease associated with heart disease as found to exist amongst the living insane ; and, as, in the majority of the cases of melancholia examined, the disease was of com- paratively short duration, I presume that in most cases (and, indeed, I know for a fact in many instances) the heart disease existed on admission, and may therefore have stood in some causal relation to the insanity, in an exciting, modifying, or predisposing manner. Though further en- quiry is decidedly necessary before one is entitled to speak with certainty on this matter, I may, at all events, before concluding, just indicate that *melancholia* (especially in its hypochondriacal forms), *monomania of suspicion,* and *impulsive* modifications occurring in *sullen* and *morose* patients, are those forms and modifications of insanity which the results of my observations lead me to suspect will most commonly stand— as effects to cause—in relation to heart disease. At any rate, it is a fact that the most intractable cases of melancholia in the West Riding Asylum are, in almost every instance, associated with very considerable heart disease, which, by its presence, is not unlikely to have caused or predisposed to the particular form of insanity in the first instance, and, by its necessary continuance, unfavourably modifies and almost renders the cases utterly hopeless. I should very much have liked to add details of the condition of the heart in all the cases of

the forms of insanity just above indicated, but must defer doing so from want of space. I may, however, state that, as a general result in these cases (including under the head of melancholia, for the present, only those cases in which it was hypochondriacal), the heart was very considerably diseased in almost every instance ; and when one just thinks what a *pabulum* for the origin and growth of hallucinations, illusions, and delusions, is to be found in the well-known subjective symptoms of heart disease, this very common association of these particular forms and modifications of insanity with heart disease is not, perhaps, after all, matter for much wonder. Further research is, however, as I have just said, required for the full elucidation of this point, and before we can speak more definitely on the subject ; and a part of this research would consist in endeavouring to ascertain if these special forms and modifications of insanity are more prevalent in those counties where there is the greatest amount of heart disease than in those where it is least. I can only, therefore, in the meantime, speak thus generally, but hope, after a more minute investigation, to be able to give more decided information on the subject.

Let me now, finally, just as briefly as possible, recapitulate and collate the conclusions at which, as the result of my observations, so far, I have arrived, with regard to heart disease and insanity :—

1. There is a remarkable relation between heart disease and insanity, in that, co-existing with the gradual but steady annual increase of the latter—both absolutely and proportionately to the increase in the population—there has been a similar and more than *pari passu* increase of heart disease throughout England and Wales, for several years past ; and the proportion of insane persons to every thousand of the population is in a direct ratio to the amount of heart disease in the different parts of the country, being greatest in those districts where heart disease is most prevalent, and smallest where it is least so.

2. Heart disease, as a *certified cause of death*, is scarcely more common in the West Riding Asylum than in the West Riding of Yorkshire generally.

3. An examination into the state of the heart of the insane, as found after death and during life, shows that heart disease, in its various forms, is exceedingly common amongst them, and, presumably, much more frequently met with in asylums than out of them.

4. This is shown by the fact that the mean average weight of the heart is considerably greater amongst the insane than the sane, and by the following facts : 1st, that in no less than 36 per cent. of 500 patients dying in the West Riding Asylum, there was more or less disease of the valves and openings of the heart and aorta, associated with more or less hypertrophy of the left ventricle ; whilst 14½ per cent. more presented hypertrophy, without valvular disease and only attributable to cerebral causes ; and, in about 30 per cent. more, the heart was affected with other forms of hypertrophy, was atrophied or fatty, or affected with other disease of less moment ; leaving only about 20 per cent. in which the heart was healthy ; and, 2nd, that amongst 680 living male patients, in no less than 44 per cent. there were physical signs of more or less heart disease, to say nothing of those cases in which the heart was weak and atrophied, or otherwise affected by disease not discoverable, in the living subject, at a single sitting.

5. More or less hypertrophy of the left ventricle was generally associated—either alone, or complicated with hypertrophy or dilatation of the right side of the heart—with the valvular disease, which existed to a greater or less extent in 23 per cent. of the cases examined. In an additional 20 per cent. of the cases, there was more or less hypertrophy or dilatation (without valvular disease), either of the right side alone or in combination with left-sided hypertrophy, more commonly the former.

6. The comparative excess in the mean average weight of the heart of the insane (amounting to 1 ounce, in each sex over the highest healthy standard average of 11 ounces for the male, and 9 ounces for the female) is attributable, in part, to the great amount of valvular disease, and, in part, to the common occurrence of hypertrophy of either ventricle, but chiefly of the right, without valve disease, observed amongst

them. This form of hypertrophy, unaccompanied by valvular disease, was found most frequently in active maniacs—chronic or recurrent—and amongst consecutive dements; and it can only be accounted for, in the great majority of cases, by the effects of the strain on the heart caused by past or present prolonged mental and physical excitement and over exertion, as well as by the obstruction to, and interference with, the systemic circulation in the brain, which arises from the minute and other obscure vascular changes in certain forms of insanity.

7. The mean average weight of the heart is considerably greater in general paralysis, and in chronic or consecutive dementia (disorganisation of the brain), than it is in other individual forms of insanity.

8. The forms of mental insanity most commonly associated with heart disease are *hypochondriacal melancholia*, that particular form of chronic mania termed *monomania of suspicion*, and such *modified* forms of general insanity as those in which the patients are *sullen* and *morose*, or *impulsive* ; and the very great frequency of heart disease in those forms and modifications of insanity justifies a strong suspicion that it is associated with them in some *causal* relation, whether it be exciting, modifying, or predisposing; the subjective symptoms of heart disease furnishing a rich *pabulum* for the origin and growth of hallucinations, illusions, and delusions. This is, however, a matter requiring further elucidation, especially with regard to the nature and symptoms of the mental insanity, when it and heart disease are co-existent *at the time of admission* into an asylum.

9. Chronic and advanced insanity is essentially of an *asthenic* type, and characterised by a greater or less languor of the general circulation, as evidenced by more or less coldness and lividity of the extremities, and weakness, smallness, or compressibility of the pulse. Such conditions can be shown to be purely cerebral or neurotic, and may be present to such an extent, in some cases, that the pulses of the extremities of the body cannot be felt, even with the most careful and prolonged manipulation. Abnormal *slowness* of the pulse—another neurotic condition of the circulation—is also not unfrequently met with amongst the insane.

NOTES

CONDITION OF THE TYMPANIC MEMBRANE

IN THE INSANE.

PART I.

By JOHN C. GALTON, M.A. (Oxon.), M.R.C.S. F.L.S.

CLINICAL ASSISTANT, WEST RIDING ASYLUM.

Introductory—Vascular Relations of the Tympanic Membrane—Method of Examination of the Tympanic Membrane—Experiments with Nitrite of Amyl—Examination of Patients, mostly Epileptics—Provisional Conclusions.

INTRODUCTORY.

IT is with some diffidence that the writer has undertaken the investigation of the subject which is somewhat crudely and superficially handled in these few pages, but he may, perchance, be permitted to offer as an excuse—first, the temptation of ploughing the first furrow in a field either as yet quite untilled, or which has been suffered for a time to lie fallow; and, in the second place, the natural desire of one who, though not a professed aural surgeon, would fain turn to some little account what he may have learnt, *inter alia*, in the ' Krankenhaus ' at Vienna, in the cliniques of Professor Gruber and Politzer.

Though the alienist may derive little or no profit from the results of examinations here recorded, touching either the diagnosis or the treatment of mental disease, and though it may be neither desirable nor probable that he will ever consider the aural speculum as essential an item in his *armamentarium* as he yet does the gag and stomach-pump, it

is, nevertheless, to be hoped that the physiologist may accept such inductive method as is here employed, either as establishing a new fact, or, at all events, as giving additional support to reasonable *à priori* deductions.

While engaged upon the work which forms the subject of this paper the writer was informed by an American correspondent that ' this has also been attempted by some German, with negative results.' The writer has, however, not as yet succeeded in obtaining either the name of this author or any reference to his work, and is further unfortunately cut off by long distance from access to any library which would contain foreign works on such a subject as Otology.

There is no allusion whatever to the condition of the tympanic membrane in the insane, either in Tuke and Bucknill's well-known work, or in its German rival by Griesinger; likewise in Gruber's treatise upon the treatment of ' Diseases of the Ear '[1] there is no mention made—as far as can be ascertained from a work of 647 pages without an index—of this subject.

Kramer's work, too, translated by Dr. Risdon Bennett, and the various papers upon aural subjects contributed by Mr. Hinton to the Guy's Hospital Reports have been consulted in vain on this subject.

In Von Tröltsch's work, however, upon the ' Diseases of the Ear,'[2] some slight mention is made of the tympanic membrane in the insane, for we are informed (*op. cit.*, p. 531) that Dr. Köppe (of Halle) found that out of 31 insane patients in his asylum who had disease of the ear, 7 had a chronic hyperæmia of the vessels of the handle of the malleus, and, besides the subjective aural sensations, aural illusions and hallucinations.

Meagre though the literature be on the subject, there is all the more reason why some observations should be carried out in this line of enquiry, in order that we may be enabled to come to some conclusions, even though they be but negative, as to whether there exists any correlation between the condition of the tympanic membrane, especially as re-

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[1] 'Lehrbuch der Ohrenheilkunde.' Wien, 1870.
[2] American Translation, 2nd edit. New York, 1869.

gards its vascularity, and any form of cerebral disorder, especially such as would give rise to some kind of insanity.

The Vascular Relations of the Tympanic Membrane.

The vascular relations of the tympanic membrane are of great complexity, but it will be the endeavour of the writer to present these in as condensed and accurate a form as possible, though, unfortunately, he has but few authorities at hand for reference—viz., the well-known Quain and Sharpey,[1] Kessel,[2] and Gruber.[3]

The blood supply of the tympanic membrane, according to the last-named author (*op. cit.*, p. 138), is derived from two quite separate arterial sources. These are—

1. A. auricularis profunda, from the internal maxillary.

2. A. tympanica—a twig of the ramus tympanicus of the stylomastoid.

Both of these are thus indirectly derived from the external carotid artery.

The tympanic artery, after being reinforced by small vessels which take origin from the deep auricular, then makes its way into the tympanic cavity *via* the fissure of Glaser.

Arrived within the ear, each of these branches takes a course perfectly independent of its previous companion—the deep auricular, now the external tympanic, ramifying only in the epithelial layer of the tympanic membrane, while the tympanic, as the internal tympanic, is solely confined to the internal or mucous layer.

Both arteries now break up into a capillary network, each in its own stratum, being separated from one another by the intervening non-vascular[4] *membrana propria*, and anastomosing only at the periphery of the tympanic membrane.

[1] 'Elements of Anatomy,' vol. ii. p. 752, 7th edit. London, 1867.

[2] 'The External and Middle Ear, excluding the Eustachian Tube,' vol. iii. p. 42, of Stricker's 'Manual of Human and Comparative Histology.' Translated by H. Power, New Sydenham Society, London, 1873.

[3] *Op. cit.*, s. 139, Wien, 1870.

[4] According, however, to Kessel (*loc. cit.* p. 42), this middle layer, as well as the cuticular and mucous layers, is provided as well with vessels as with lymphatics and nerves.

The cuticular, or outer, layer of the tympanic membrane ·
has the richest capillary network. From the roof of the
meatus there descends a single—in exceptional cases two—
arterial twig upon the membrane, and there also pass other
small arterial twigs from the sulcus tympanicus over its
surface, following the whole extent of its periphery. The
branch just described as descending upon the membrane
from the roof of the meatus runs downward along the outer
edge of the handle of the malleus until it reaches the centre
of the membrane, giving off all along its route numerous
branches in a radial direction, and becomes finally continuous
with a capillary network which is in relation with the delicate
vessels which enter from the sulcus tympanicus.

The vessels of the internal, or mucous, layer of the tympanic
membrane are much more delicate. According to Gerlach
there is a distinct capillary network, to which the internal
tympanic artery chiefly contributes, and which runs down
parallel to the handle of the malleus.[1]

According to Kessel (*loc. cit.* p. 42) the artery just de-
scribed as descending upon the handle of the malleus from the
roof of the meatus, after crossing the lower extremity of the
manubrium mallei, ' divides into two branches, of which one
supplies the anterior inferior quadrant.'

' The branches running in a centrifugal direction in the cutis, and here
and there connected by transverse or oblique anastomoses, terminate in
capillaries, which on the one hand unite to form the smaller veins accom-
panying the arteries, and on the other hand pass straight into the venous
plexuses, of which one encircles the manubrium, and conveys its blood into
the posterior superior veins of the cutis of the auditory meatus, whilst the
other lies at the border of the membrana tympani, and likewise conveys its
blood in an outward direction.'

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[1] Gerlach states (see Gruber, *loc. cit.* s. 139) that he once succeeded in demon-
strating that the vessels of the mucous layer are isolated, for after injecting a
brain through the two internal carotids, after ligation of both vertebral arteries,
he found the internal tympanic plexus full. This was due, he thought, to an
anastomosis between the internal auditory artery (a branch of the basilar), and
the arteries of the tympanic cavity. According to Langer, however, the internal
carotid itself gives off delicate shoots to the mucous membrane of the tympanum,
so that it is probable that the result of Gerlach's experiment was rather due to
direct injection of the internal tympanic plexus, *viâ* the internal carotids.

The capillary plexus, which lies centrally in the membrana propria, communicates with that of the cuticular layer externally, and internally with that of the mucous lining.

The internal plexus, that of the mucous membrane, essentially a capillary one, is distributed around the manubrium and about the tendinous ring, the plexus around the latter being a prolongation of the capillaries of the mucous membrane of the tympanic cavity. The blood of this membrane is then carried off in two ways—by the veins of the tympanic cavity, and by those of the external auditory meatus.

'The chief proportion of the blood traversing the arteries of the membrana tympani and the capillaries may therefore enter the larger veins by very different routes; by a shorter path, into the plexus of the malleus, and by a longer path, over the membrana tympani into the marginal plexus. The path traversed by the blood during life will obviously depend upon the nature of the mixtures which it unites in the different veins. It may, however, be said with certainty that the arterial blood always returns by the shortest route through the plexus around the manubrium, where no special obstacle is presented to its course in the veins into which the vessels of this plexus discharge themselves.'

The auricular veins, with which some of the vessels of the internal plexus communicate, are tributaries of the temporal vein, and are thus in indirect communication with the external jugular.

The veins of the tympanic cavity, with which the marginal part of the same plexus indirectly communicates, run to join—

a. A venous plexus in the neighbourhood of the mandibular articulation, and thus communicate with the internal jugular vein.

β. The pharyngeal veins, and then communicate directly or indirectly—through the medium of the superior thyroid or lingual vein—with the internal jugular.

γ. The middle meningeal veins (*mittlere Hirnhautvenen*), which meet the internal maxillary, and are thus brought into relation with both the internal and external jugulars.

From this analysis, which it would be difficult further to condense, it will be seen that however intimate, though complex, be the vascular relations *inter se* of the various layers of the tympanic membrane, such relations, as regards the arterial

and venous systems within the cranial cavity, can be but at most termed indirect.[1]

METHOD OF EXAMINATION OF THE TYMPANIC MEMBRANE.

The examination of the tympanic membrane has this advantage over that of the fundus oculi, that we have here to deal with a fixed and stable surface, perfectly insensible to light, while, on the other hand, he who uses the ophthalmoscope must take his chance of catching the ever-fleeting image which is presented to him by an organ in perpetual movement, and highly sensitive to the strong light which must of necessity be used for its examination. The examination of the tympanic membrane has, moreover, this further advantage, that it may be, is, in fact, preferably conducted in a room illuminated by diffused daylight, while, for a sufficient view of the interior of the eye, powerful and properly adjusted lamplight and a completely darkened chamber are absolutely essential.

Apart, too, from its convenience, the daylight method of illumination is obviously preferable, in that the real colour of the tympanic membrane, and of the several objects in relation with it, is masked by no fallacious yellow tint, such as is intruded in an examination by artificial light—an advantage which the writer had opportunity of appreciating by comparative observations carried on in the artificially illuminated salon of Professor Politzer, and in the contracted crowded closet, lighted only by two small windows—the translucency of whose panes was further diminished by the crystals of daily frosts—which served as the clinique of Prof. Gruber.

The examination of the tympanic membrane has, however, this disadvantage in comparison with that of the fundus oculi, that it is too frequently—in more than one third of the cases which the writer has examined—hindered by the blockage

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[1] With regard, however, to the auriculares posteriores, branches of the external carotids which partly supply the pinna with blood, Schroeder van der Kölk states that the vertebrals, before they enter the cavity of the skull, give off muscular branches which anastomose with these arteries, and from this explains why in intracranial congestions, especially such as have their expression in epilepsy, he has found repeated cuppings in the neck act favourably.

of the external meatus with a firm bung of cerumen, which can frequently only be dislodged at the sacrifice of much time and patience.

As the object of the research which forms the subject of this paper was solely to ascertain the vascular condition of the tympanic membrane, with especial reference to the appearance of the vessels which run upon the handle of the malleus, such variety of instruments as would be needful for the investigation of the state of the internal ear, and for the exploration of the tympanic cavity and Eustachian tube was, of course, not needed.[1]

For the following reasons, too, the writer did not apply himself much to the use of the ear-syringe. As the ' Material ' —as a German would say—in the asylum was so plentiful, it was found more convenient to at once discard the patient whose ears were blocked up by cerumen than to expend much time and patience upon attempts to dislodge the obstruction ; for, were this effected, a fallacious congestion of the vessels which run upon the handle of the malleus might have been presented to the observer, due to the continuous pressure of the wax plug upon the tympanic membrane.

All the instruments, then, which the writer found absolutely necessary were very few in number, and such as could easily be carried in the pocket—viz. a Tröltsch's reflector—a concave mirror, three inches in diameter, and of six inches focal distance, mounted upon an ebonite handle, and resembling the reflector used in laryngoscopy in that it has at the centre a small circular space, free from amalgam backing, through which the observer looks ; and three funnel-shaped German-silver specula, of the three usual graduated sizes, and of the circular pattern, as used by Prof. Politzer.[2] Kramer's speculum is not to be recommended, as not only do the hairs which so frequently thickly clothe the wall of the meatus have a tendency to fall between the blades of the instrument, when these are drawn apart, and thus obstruct the view, but the

[1] *e.g.* Tuning-fork, otoscope, Eustachian catheter, Politzer-bag, &c.
[2] The writer would have preferred the oval pattern, as recommended by Professor Gruber, but the instrument maker, either not having this in stock, or presuming upon the supposed ignorance of his customer, sent the Politzer pattern.

very introduction of the instrument may be rendered painful to the patient by a forcible and sudden dilatation of the walls of the meatus to which the observer is very prone to resort.

The patient having been seated in a chair, parallel to a good-sized window, the observer introduces, with his *right* hand, the speculum a short distance within the meatus of the ear to be examined. The instrument is next held between the forefinger and thumb of the *left* hand, while, in order to bring the cartilaginous and osseous portions of the meatus into the same straight line, the pinna is simultaneously grasped and lifted upwards, and somewhat backwards, between the middle and forefingers of the same hand, the tip of the forefinger resting in the fossa of the antihelix, and still, in combination with the thumb, controlling the course of the speculum.

The reflector is then taken into the right hand and the light thrown upon the tympanic membrane, the reflected rays from which reach the eye of the observer through the circular window at the centre of the instrument.

The above directions, though they may seem absurdly minute, may, if followed out, save the patient's ears from much unnecessary pulling about, and the operator some clumsy and fruitless fumbling.

As this paper does not profess to be a treatise on aural surgery, a very brief description of the land-marks of the tympanic membrane must suffice.

On looking through a speculum placed, say, in the right ear, we see an irregularly circular, membranous-looking surface, of a pearly-grey tint, and of about the area of a three-penny bit. Next, running from above obliquely downwards and backwards, as far as the centre of the area, will be seen a yellowish-white sabre-like body, slightly curved at the tip. This is the handle of the malleus. (*Manubrium mallei.* 'Hammergriff,' *Germ.*)[1] Quite at the upper and anterior

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[1] Sometimes erroneously termed the 'long process' (*processus gracilis*). The real 'long process' is, however, a rudimentary remnant—situated between the *processus brevis* and the head of the malleus—of a structure which, as 'Meckel's cartilage,' was continued along the ramus of the mandible during fœtal existence.

extremity of the handle of the malleus, there is always visible, with varying degree of distinctness, a small round ivory-like knob, of about the size of the point of a knitting-needle.   This is the *processus brevis* of the *malleus* ('kleiner Fortsatz,' *Germ.*).   Stretching away anteriorly and posteriorly from this little knob are two folds in the tympanic membrane —the 'Taschenbänder' of the Germans—the posterior of which is usually most distinct, which vary in prominence according to the obliquity of the handle of the malleus. Lastly, radiating from the tip of the *manubrium mallei*, downwards and forwards to the periphery of the membrane, there will be seen, making allowance for various modifications in shape, a conical streak of light ('Lichtkegel,' *Germ.*), having its base at the circumference of the membrane, and its apex at the tip of the handle of the malleus.   This cone is of great importance as an index of the degree of tension of the tympanic membrane, and of the direction of its bulging.

The area of the drum-head may be, for purposes of description, conveniently mapped out, vertically by the handle of the malleus, and horizontally by the upper boundary of the 'Lichtkegel,' into quadrants, which may be termed the anterior superior and inferior, and posterior superior and inferior quadrants respectively.

It should perhaps be mentioned that, owing to the occasional transparence of the tympanic membrane, the downward process of the *incus*, which articulates with the *stapes*, is visible through the posterior superior quadrant, and that the *promontorium*—a projection upon the inward wall of the tympanic cavity formed by the first turn of the *cochlea*— dimly shines through the space anterior to the handle of the malleus.

The following abbreviations have been found by the writer convenient for purposes of note taking, and will be employed in his record of cases :—

<div style="text-align:center">

R E   Right ear.
L E   Left ear.
 P   Pinna.
t m   Tympanic membrane.

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m m	Manubrium mallei.
p b	Processus brevis.
t b	'Taschenband.'
l k	'Lichtkegel.'
p m	Promontorium.
i	Incus.
a s q	Anterior superior quadrant.
a i q	„ inferior „
p s q	Posterior superior quadrant.
p i q	„ inferior.

The diagnosis of perforations, scars, calcareous concretions, &c., &c., does not come within the province of this paper, but it may be as well to hint at the possibility of mistaking for a hæmorrhagic patch a tiny speck of cerumen—perhaps temptingly lodged below the posterior 'Taschenband' of the tympanic membrane, at the bottom of an apparently perfectly clean meatus—a fallacy which on one occasion so far led the writer astray that he made a careful sketch of the supposed hæmorrhage. As, however, this deceitful εἰκων disappeared at the persuasion of a powerful syringe, the drawing, instead of being made public, will merely be retained as a wholesome warning against future error.[1]

As the observer is very liable to mistake a portion of the wall of the meatus for the tympanic membrane, he will find it a convenient rule first to make search after the bead-like projection of the *processus brevis* of the *malleus*. This landmark being found, all the other structures directly or indirectly in relation with it may then easily be mapped out.

EXPERIMENTS WITH NITRITE OF AMYL.

After the investigation from the side of anatomy as to whether there exists some connection between the vascular condition of the tympanic membrane and the parts which lie within the cranial cavity, it would seem philosophical

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[1] *Apropos* of hæmorrhagic spots, it may be worth while to mention that these are capable of a sort of migration, probably owing to the mutual attrition of the two layers of tympanic membrane between which they may be effused. The writer has now before him a sketch of the wandering to the extent of a quarter of an inch in an upward direction of a drop of blood resulting from a paracentesis ('myringotomy') performed upon a patient by Professor Politzer.

were we to attempt to approach this question from the side
of physiology. The pathological side of the question is the
subject and end of this paper, and may, it is trusted, be the
means of shedding in turn upon the physiological side some
small glimmering of light.

We have fortunately an agent by means of which we may
rapidly influence the circulation, through its inhibitory
action upon the vaso-motor centres, and thus cause at will
a temporary hyperæmia of those parts especially which lie
at the upper part of the trunk. This agent is nitrite of
amyl.

Into the question of the general and special action of this
drug it is not my province to enter. Full information on this
point will be found in Dr. Crichton-Browne's paper on
'Nitrite of Amyl in Epilepsy,' in this number of the 'Reports
of the West Riding Asylum,' and in Dr. Aldridge's paper on
' The Ophthalmoscope in Mental and Cerebral Diseases,' at p.
97 of the first volume of the same series of reports. The
latter observer, to judge from nine experiments which he
describes in detail (*loc. cit.* pp. 95–97), found invariably, after
the nitrite had been inhaled, that there was an evident
increase in size of the retinal arteries, accompanied by a
deepening of the capillary tint of the disc, while little or no
change was visible in the calibre of the veins.

Let us now see what results—as regards the vascular con-
dition of the tympanic membrane—we gain, after the inhala-
tion of the nitrite, in 35 cases.

All of the 35 persons who inhaled the drug were males. Out
of these, 15 were sane persons, mostly attendants, 16 were
epileptics, while the remaining 4 were patients suffering from
other kinds of mental disease.

In all of these cases the drug exercised externally and
rapidly, often in much less than a minute, marked effect, some
of the patients, though averse to inhalation at first, taking to
it ' very kindly,' and becoming hilarious and much elevated in
spirits. It should be stated that in all the cases selected for
experiment there was no previous hyperæmia of the vessels
upon the handle of the malleus.

After the inhalation of the drug some congestion of the vessels was seen in 10 cases, out of which the effect was well marked in 6.

Of these 6 were epileptic,

        3 sane,

        1 affected with monomania of suspicion and hallucination of hearing.

Of the 6 epileptics, the congestion was well marked in 3, while there was also well marked congestion in 2 out of the 3 sane persons and in the case of monomania.

The 6 experiments in which the congestion was well marked may be described a little more in detail :—

EXPERIMENT I.—H. B—. Epileptic, taking bromide of potassium, stripped to the waist. After inhalation of the nitrite, considerable blushing over face, ears, arms, and chest. *R. E.* Marked congestion of vessels over *m m*, which, with the *t m*, was decidedly pale before the administration of the drug.

EXPERIMENT II.—C. O—. Epileptic, taking no medicine, also stripped. Effects of drug externally evident. *L. E.* Before experiment *m m* pale, no vessel being visible. After inhalation, distinct congestion, especially below the *p b*.

EXPERIMENT III.—J. K—. Epileptic, taking bromide of potassium. *L. E.* Before inhalation *m m* pale, vessels being but faintly visible. Well marked congestion after inhalation.

EXPERIMENT IV.—A—. Attendant. *R. E.* Vessels over *m m* just visible before inhalation. Afterwards most decided change. Second observation five minutes later. Congestion persistent.

EXPERIMENT V.—P—. Attendant. *R. E. p b* rather prominent. Vessels just visible. After inhalation, very well marked congestion.

EXPERIMENT VI.—T. S—, taking no medicine, monomania of suspicion, with hallucination of hearing. *R. E. m m* drawn very much inwards, so that the *p b* projects considerably. (This is also the case in *L. E.*) Vessels just visible. After inhalation marked and decided congestion.

It must, however, by no means be assumed that a turgescence of the vessels of the face, or even of the tympanic membrane is of necessity an index of an equivalent hyperæmia of intracranial parts. The late Professor Schröder van der Kölk [1] makes the following very pertinent remarks on the subject :—

[1] 'The Pathology and Therapeutics of Mental Diseases,' translated by J. T. Rudall, pp. 33, 34, London 1870.

'The spreading also of the vessels in the face conduces to derivation from the brain. There are not many other arteries which are so easily filled with blood and extended as the arteries of the face : they are everywhere surrounded by soft fat, and on that account the coats of the vessels have no powerful support from without. Thence it occurs that the cheek, and indeed the whole face, so easily assumes an increased colour, e.g. in powerful and sudden emotion, especially also from high degrees of temperature. If the vessels of the brain could be as easily filled, we should have to fear an apoplexy from every rather active exertion. But the carotis externa thus leads the strong pressure of blood outwards, because the carotis cerebralis in the closed cavity of the skull cannot equally well expand, and on that account opposes a more powerful resistance to the pressure of blood. Therefore, from diffused redness of the face, we may not always conclude that there is congestion of the brain, though the latter is frequently associated with the former.'

Examination of Patients, mostly Epileptics.

In addition to the 35 cases experimented upon with nitrite of amyl, the writer has examined the ears of 234 persons in the West Riding Asylum, the great majority of whom were patients, mostly epileptic.

Out of these 234 examples, 145 were males and 89 females.

Of these 27 persons were sane, 22 being males, and 5 females.

The remaining 84 females were epileptic patients.

Of the remaining 123 males, 101 were epileptics, and 22 patients affected with other forms of mental disease, out of which, 7 were suffering from melancholia, and 7 from general paralysis of the insane.

Out of the 89 females,

The tympanic membrane was not seen in either ear, owing to cerumen, resistance on the part of the patient, &c., in 36 cases.

The tympanic membrane was visible on both sides in 36 cases.

The tympanic membrane was visible on one side only in 17 cases.

Out of the 84 female epileptics, the tympanic membrane was seen in 48 cases.

Out of these 48 cases, congestion of the vessels running along the handle of the malleus was noted in 19, that is, in about five-twelfths.

The congestion was seen in both ears in 7 out of these 19 cases, in the right ear alone in 4 cases, and in the remaining 8 cases in the left ear only; but in 2 out of these 8 cases, only the one ear was available for examination.

Out of the 101 male epileptics, the tympanic membrane was seen in 58 cases.

Of these 58, in only 17 was there more or less congestion of the vessels upon the handle of the malleus.

Out of these 17, the congestion was seen in both ears in 6 cases, in the right ear only in 4, and in 7 in the left ear alone.

Of the 17, 14 were taking no medicine at all, while the remaining 3 were being treated with bromide of potassium.

It will thus be seen that out of 106 epileptics, of both sexes, in which the tympanic membrane was seen, more or less congestion of the vessels running over the handle of the malleus was observed in 36 cases, or a little over 33 per cent.

In none of the 27 sane persons examined was any hyperæmia of the vessels upon the handle of the malleus observed.

Some attempts made to ascertain the condition of the vessels of the tympanic membrane in epileptic patients immediately after a fit, were baffled by the unfortunate coincidence of the presence of a plug of cerumen in the meatus.

The writer has not as yet succeeded in obtaining a view of the tympanic membrane in ears afflicted with the characteristic blood-tumour, 'Othœmatoma.' It would be interesting to establish some correlation between a recognisable vascular change in the tympanic membrane and a morbid product which, be it brought about spontaneously or by violence, points towards some peculiar condition of the circulation in the neighbourhood of the ear.

PROVISIONAL CONCLUSIONS.

From the experiments and observations above recorded, it may be concluded :—

1. That when a change in the vascularity, e.g. hyperæmia, of parts both within and without the head are simultaneously brought about by the action of a certain drug, similar changes may be recognised, but not invariably, in the vascularity of the tympanic membrane.

2. That with certain cerebral disorders, e.g. epilepsy, such as would tend towards, or be produced by a hyperæmic condition of parts at the base of the cranium, there is sometimes, but by no means invariably, correlated a hyperæmic condition of the vessels of the tympanic membrane.

3. But that many more comparative observations upon the sane and the insane, and further experiments as to the action of drugs, such as ergotin, atropin, &c., which are known to exercise a specific action upon the vaso-motor system, are required, before that such conclusions will have much value, either physiologically or pathologically.

OBSCURER NEUROSES OF SYPHILIS.

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When, in obedience to the kind insistence of my friend Dr. Crichton Browne, I undertook to write upon the present subject, I was scarcely aware that what were to me the obscurer neuroses of syphilis are in all likelihood less obscure to more instructed observers. On proper research I now find that there are few of the disorders I may put under this head which have not already been described with more or less completeness by such eminent 'syphilographers' as Sigmund, Gros and Lancereaux, or Ricord. Nevertheless I deemed it right to pursue my first intention, for I am disposed to think that certain manifestations of syphilis which, as such, were unfamiliar to myself, are likewise unfamiliar in greater or less measure to other physicians ; and I may therefore do well to call attention to a somewhat neglected order of facts which are not curiosities, or not mere curiosities at any rate, but which are, on the contrary, of the greatest practical importance both in a clinical and in a therapeutical point of view. Moreover I think that the fascination which the discovery of obvious pathological changes has exercised, and will always exercise, over us, has thrown into the background those syphilitic affections which are not attended by such obvious or discoverable changes.

By the obscurer neuroses of syphilis I mean those which depend, so far as we can tell, upon tissue changes which are not obvious, or to be discovered by the microscope, and

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which we call functional. They may be roughly classified
as follows :—

(1). Intellectual and emotional disorders, such as dementia,
depression, fear, melancholia, delusion, morbid susceptibility,
irritability of temper, ' loss of tone ' and so forth.

(2). Sleeplessness.

(3). Motor defects, such as tremor and muscular debility—
perhaps epilepsy.

(4). Neuralgia, occurring in both superficial and visceral
nerves.

(5). Nutritive defects, such as anæmia, which do not
belong to the neuroses, may here be dismissed with the
remark that anæmia may exist alone as a symptom of
syphilis, and may clear up with strange rapidity on the
administration of mercury. Few physicians, perhaps, are
not familiar with this result.

Now it is open to any one to object, and to sustain the
objection, that changes of the kind I have reckoned up can-
not be ' immaterial,' and must, as a fact, depend upon some
such change as proliferation of connective elements either
within or upon the nerves or ganglia concerned.

As a fact, indeed, certain recent observers have described
such marks of irritative action in nerve strands, in sympa-
thetic ganglia and other places,[1] but I still think that even if
their statements be verified, there is no such change in the
cases to which I more especially refer. The phenomena of
which I speak are often fugitive or unsettled in position,
and when cured, if they relapse, it is often under new forms.

Their behaviour is unlike that of optic neuritis, which we
can watch, and unlike neuritis of the third, sixth, or other
cranial nerve, the state of which we know pretty well by
inference. To them the mischief clings, and it recurs as
often as it is expelled, so that he is a lucky patient who,
after years of watching, can say he has no more fears. I
much regret to have to say that my own experience of
positive nerve lesions in syphilis is much less comfortable
than that of others would seem to be. Promptly they may
recede under appropriate remedies, but they recur again and

[1] Vid. e. g. Petrow. Virch. Archiv. xvii. March 1873.

again, to be driven out less and less easily. I lately looked through the very numerous notes of syphilitic disease of the nervous system in my casebooks, when preparing this paper and I was really shocked to find how few even of my most successful cases had survived. Within a very few years the recurring mischief, if it has been at all severe, grows to such an extent that it can no longer be controlled, and the patient sooner or later succumbs to it.

But in the cases to which I now refer recovery is the rule, and by this I mean recovery as complete as that which follows the proper treatment of an ordinary dyspepsia; with a little care and patience we may almost promise the patient that he shall be cured, and the cure is generally as permanent as it is easy. A course of mercury, gently prolonged, a month at Aix, or large doses of sarsaparilla, will rarely fail to dismiss the evil for once and all, so far, at least, as that particular manifestation is concerned.

In fact a syphilitic tic, gastralgia or melancholia, is easier to cure than these affections prove to be when due to other causes; and yet we scarcely suppose that in these we have to deal with any massive dislocations of the tissue elements. Between the words functional and structural, as applied to disease, there is an appearance of antagonism which of course is false, but this false appearance serves well, if its ultimate fallacy be borne in memory, to indicate a clinical distinction which is true up to a certain point. This distinction does not lie in the transient character of the affection, for a common tic may be more difficult to cure than one which depends upon an injurious irritant capable of removal.

Talking, as we must often do, in terms which are not defensible in the extreme, we thus signify that a part suffers not because of any massive changes in its tissue, but because of some change in its molecular movements or nutritive currents. These molecular deviations by accumulation may become massive, but clearly there is some latitude of molecular vibration within which equilibrium is not endangered, and such vibrations do suffer modification from any one of many causes, such as cold, certain qualities or quantities of blood, certain inherent defects of tension, certain external irritants

and the like. To transmit normal impulses in a normal way, the molecular vibrations must probably have a certain value —must take place x times per second—and any increase or diminution of this means a less delicate balance, and less susceptibility, therefore, to outer impressions. Hence the curious similarity, as regards function, between an oversensitive, or rather an overfelt nerve, and one that is benumbed. In syphilis, then, we have to do with two kinds of nerve pain.

1. That due to something more than perturbed molecular vibrations, namely, to a positive dislocation of the tissue elements by irritative proliferation, which, however, is not in the nerves at all, but in the connective tissues lying near them.

2. That which is a primary neurosis in a stricter sense, being from the first a perturbed rate of molecular vibration in the nerves themselves, which is probably attended by correlative vascular changes, but which, so long as it does not exceed certain limits, is compatible with structural integrity, as a salt may be heated to a given point without decomposition. It is the first kind of syphilitic nervous disease which of late has received the most attention ; but it is of the second kind I now wish more especially to speak. How syphilis can thus act directly upon the nutrition of the nerves is as yet beyond the scope of direct enquiry ; but that it is not by way of mere impoverishment of the blood is pretty clear from the fact that those measures which tend simply to improve the globulation of the blood are ineffectual to remove the morbid phenomena. Nor are those ' nerve tonics ' such as arsenic, zinc and the like, any more successful, while on the other hand the most obstinate sufferings will vanish like a cloud before a few doses of mercury or iodide of potassium. Hence the importance of an etiological diagnosis.

I shall now proceed to cite some illustrative cases, which may give rise to farther comments as we proceed. The cases are taken entirely from my own books, with the exception of some few which have come before me at the Infirmary. I am compelled, however, to omit many details which I fear might lead to the identification of the patients concerned. Of the obscure syphilitic neuroses, none are more common than

the mental (1), and I am disposed to think that symptoms of this kind often remain uncured for lack of knowledge of their true nature. Take for instance the following. I met a gentleman in a railway carriage who had consulted me some three years previously for secondary syphilis, and I naturally enquired after his health. He looked well, but surprised me by the despondent nature of his replies. He said that he was on the point of consulting me again (which he did in fact a few days later), as during the last few weeks he had felt most keenly how horrible a disease this syphilis is, how disgraceful it is to an honourable man and a gentleman, and how destructive to his constitution. These were platitudes to which I could hardly withhold my assent, but the expression of them in my patient's mouth created some transient wonder in my mind. As I saw him again and again, I was satisfied that the patient was under the influence of a morbid mental condition, and I questioned him carefully as to his family history, which however is excellent. He was himself the very last of men to hold these views. He was and is a very successful man of business, a keen sportsman, vigorous and sane in mind and body, and of that cynical character which is wont to see too little rather than too much in sexual sins and their results. But he said he had now for the first time fully realized their true bearing on society and upon himself in particular, and was in the depths of shame and despair. I talked in vain, and I sought in vain for spots or outward symptom; I prescribed change, which made him worse, and for weeks I failed to give him the least relief by tonics or sedatives, until it occurred to me to put him through a course of specific treatment, as he had some nocturnal pains and a sallow face. I need not delay the reader by any farther detail, suffice it to say that as he passed under the influence of mercury and iodide of potassium, his morbid fancies were dispelled, and his mental conceptions underwent a transformation which surprised even himself. I have not seen him now for six months, but I believe that he remains well and cheerful.

Take again the following:—A gentleman of about thirty years of age, a good scholar, a man of the world and

easy in temper, surprised me, who am an old friend of his, and surprised himself and his family no less, by suddenly discovering that he was harassed and depressed by religious difficulties. He positively lay day after day in bed having the Bible read to him, and reproached himself bitterly in that he had ever allowed himself to speculate upon the origin of myths or upon the incrustations of human error. In a word his whole mental attitude was changed as completely as that of the modest young lady, who, under the influence of mania or chloroform, discovers a surprising familiarity with the language of the streets. I was utterly bewildered and felt very uneasy about it, and felt still more uneasy when the whole 'platform' was gradually dissolved and a new one set up which mainly consisted of sexual subjects. This new phase was particularly painful to me, and, I must add, to himself likewise. He would allow some horrid sexual delusion to get possession of him, and, knowing its falsity, would strive in vain to rid himself of it. Meanwhile he lost all energy and all manliness of character, and I feared much he was a ruined man and would end in an asylum.

All this, which I shortly narrate, occupied some three or four months, when it occurred to me to put him under specific treatment. He was sallow, pining and slightly subject to scattered anomalous eruptions, and I had treated him for secondary syphilis two or three years before. To my surprise and pleasure his complexion improved, he gained flesh, the clouds and delusions lifted off his mind, and three weeks later he was a new man. Up to this time all treatment had been unavailing and almost useless. It is well to note that in this case, in the preceding, and in many other cases of syphilis in which neuroses, obscure or obvious, are seen, the secondary symptoms of the malady were slight. The phenomena I have classed under the first head preserve a great similarity in character: depression, fear, irritability, melancholy, delusions, are among the commoner forms of mental disorder I have myself had to treat, although I believe that anger and maniacal states have been noticed by others. I need not, therefore, multiply my own cases. I have notes of two instances in point which occurred in women, but I think it

better to omit them, as the female breast is liable to gusts of exaggerated feeling and to failure of spirits and depression which make it less easy to follow the precise working of one morbid influence. I may, perhaps, give one more example from the male sex. A gentleman, who to my knowledge had given syphilis to his wife in quite a light-hearted manner some few years before, wrote to me a while ago in words of anguish to come to him at once. In his note he described at large the traitor he had been to his family, all of which I knew well, and which he knew well that I knew. On my arrival I found him cast upon a sofa, his face buried in his hands and his voice unmanageable or inaudible. I anticipated some new turn of affairs for which his note had been the preparation. Some scenes with his wife, or, worse still, with his mother-in-law, or other dreadful event which was beyond all guessing : I was surprised, therefore, to hear nothing but the old story again, which was threadbare enough to have left him very cool on all previous occasions. My first movement was one of concealed contempt, for I believed he had been drinking ; farther watching, however, removed this suspicion, and the concurrence of some other syphilitic accidents led me quickly to the true diagnosis, for this case was not among the earlier cases in my experience.

For a week or two the symptoms remained in greater or less force ; he was depressed, full of self-reproach, fearful of impotence and so forth, but under the use of Lee's baths, with iodide of potassium and sarsaparilla, he made a good recovery in a far shorter time than the patients of which I have spoken before. In those cases of established mental disease, of which syphilis is the first cause, it is often hard to say whether positive injury to the tissues does or does not exist. For instance, a married woman, Mrs.——, has been twice in the infirmary as a patient under my care. Her history and aspect are distinctly syphilitic. On admission we found her on both occasions to be dull of apprehension, very much depressed in spirits, mute when spoken to, and indeed rarely saying a word under any circumstances. She would lie days together in bed with her face to the wall, as if all social instincts and habits were dead in her. When

she first came she was rapidly cured by specific treatment, and on the second occasion, although her progress was less satisfactory, she became convalescent in a few weeks.

In her case, however, and in many like her, the recurrence of the same symptoms in the same way, the marks of permanent mental deterioration, and the imperfect return of saner views of things, make it probable that we have to do with some chronic thickening of skull and membranes.

2. The sleeplessness of syphilis is a very curious symptom, and it is one which I at least long failed to recognize in any adequate way. During the last four or five years, however, I have understood it better and have met with it very frequently in patients whose general condition was not such as to make the diagnosis of the easiest. In children, by the way, the symptom was partly known to me, and probably to others still better, but I had not extended the same observation to adults. Syphilitic children will lie sleepless, and often will cry day and night for a week at once, so that it is a marvel how they survive the loss of rest. In many cases there is no evidence whatever that they are in pain. So it is with adults. A young man who has suffered from primary syphilis but a few years ago, or an older man whose chancre dates back ten or fifteen years ago, may come to complain that his nights are sleepless, or nearly so. He has no pain, he is warm, he has no mental strain or anxiety, his stomach is not empty, and yet after one or two hours sleep he awakes with eyes as wide as a daisy at noon. Nothing that he can do will bring back his slumbers : he makes water, paces his room, bathes his face, eats a sandwich, reads a sermon, turns resolutely to the wall—all is in vain, and night by night he fails to get sleep, his strength fails, he becomes restless and irritable by day, his flesh falls, and his condition, at first fair enough, becomes really alarming. In all these cases of causeless insomnia syphilis is to be suspected, and specific treatment will bring the rest which defied all the opiates in the Pharmacopeia. The instances I have noted of this are numerous, for sleeplessness has very commonly entered into the list of symptoms detailed by persons whose complaint included many others of a more obvious nature. In one case

where the insomnia existed alone, we failed utterly to procure sleep: medicines, changes of scene and thought, all failed to bring relief, until, fortunately, some rupial eruption appeared upon the person, and eruption and sleeplessness fled alike under the use of mercurial inunction and sarsaparilla. A very few weeks later a gentleman consulted me, for the same symptom, who occupies a high position in the church and who deserves his high position. The usual remedies failed : my former experience came to mind, and I ventured to ask him if he had ever suffered from any venereal poison. With honourable frankness he confessed that as an undergraduate he had passed through the usual phases of the malady in a mild form, and I made out that he had not been free from passing symptoms in later years. The usual specific treatment relieved him of his sleeplessness and of many other little ailments, chiefly neuralgic, the nature of which had been unknown to him.[1]

3. Of the motor defects I forbear to speak at any length, partly because I would here curtail a paper which is growing too long, and partly because the symptoms here referred to may often be but signs of incipient mischief of an organic kind. Local twitches, cramps or tremors must always be regarded with eyes of great suspicion, though I believe they are often indicative only of temporary changes. It is hard to prove what nevertheless I believe, that epilepsy due to syphilis may not in all cases be due to positive disease of the nervous centres. I have watched several cases in which epilepsy of the ordinary bilateral kind, occurring in persons having a syphilitic history, yielded to treatment by mercury and iodine, and did not recur. The very nature of these cases, however,

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[1] These cases of syphilis recurring in the after lives of responsible and distinguished persons are peculiarly distressing, and at times it is hard to enquire properly into the original cause. I have in my note-book the details of another case also in the person of a clergyman distinguished both for his abilities and for the charm of his bearing and high character ; to him, of course, the knowledge of his affection is peculiarly saddening. I need not describe the case, as in its main features it closely resembles that of others in the appearance of mental depression, sleeplessness and neuralgia cured by iodides and mercury. I allude to it to show how careful we must be not to allow circumstances to conceal from us the true nature of such cases.

prevents an appeal to the post-mortem table, and I am not
justified, therefore, in laying any great stress upon them.

I must hasten on now to the last of the heads under which
I have chosen to write, namely to the neuralgias. 4. I
believe that a very large number of syphilitics suffer from
pure, uncomplicated neuralgia, both visceral and super-
ficial, in which there is no reason to suppose that any posi-
tive lesions exist in or upon the nerves concerned. They are
not often fixed, and they do not return in the same place ;
perhaps the pain may change its place even at each paroxysm
—often the patient himself can scarcely define their position.
Again, the places where they occur, when superficial, are not
tender to pressure, as is the case in periostitis ; nor, again,
is the pain especially nocturnal nor aggravated by warmth.
For instance, Mr. —, aged 27, came to me with a severe,
wearing cervicobrachial neuralgia, which had resisted treat-
ment for some weeks.   He had a tolerably obvious syphilitic
cachexia, and a decided history of the complaint.   When
treated by the usual remedies for this disease the neuralgia
cleared off.   Another gentleman, of singularly active and
vigorous habits, and one of a very vigorous stock, came to
me with intense headache of some weeks' duration, which
he believed, and correctly believed, to be syphilitic.   But
there was no nocturnal periodicity, no tenderness of scalp,
no periostitis, and the pain once cured by anti-syphilitic
remedies has not recurred during the five or six years which
have subsequently elapsed.   In another instance a tri-gemi-
nal neuralgia had resisted every device which an exhaustive
knowledge of the therapeutics of neuralgia had suggested to
an eminent practitioner, including hypodermic morphia.
The patient came to me because he had heard that I dis-
approved of the long-continued use of that remedy, and it
so happened that I became aware of the history of a vene-
real infection.   Having then a less suspicion of the obscure
neuroses of syphilis than I have now, I nevertheless thought
it wise to administer the biniodide of mercury, and in a few
days the neuralgia was gone, and the patient's sallow, worn
look, which may have been due perhaps to the pain only,
gave place to a healthy complexion and aspect.   More

commonly, however, the syphilitic pains, neuralgia or what you will, are fleeting. Pains course over the scalp, pierce the ribs, scrape the shins, drag at the shoulder-blades and loins, coming and going like the sufferings of Falstaff when he was pinched by the fairies, or they shoot vividly from point to point, making the patient cringe too late, for they are gone almost before they are felt. These, which I presume can only be neuralgic pains, are very characteristic of syphilis, and are dissipated by the appropriate antidotes. Visceral neuralgias are far from uncommon, and of these palpitation and gastralgia are the most common varieties. A gentleman who consulted me a few months ago undoubtedly owed to syphilis a prolonged wearing pain in the pre-cardial region, attended with cardiac disturbance. His heart was quite healthy so far as its structure was concerned, but its action was irregular and occasionally intermittent. He suffered dreadfully, so he said, from palpitation, and this was brought on less by exertion than is usually the case in mere anæmia. When sitting still his heart would throb into his mouth, and when in bed his heart would flutter as if his chest was too small to contain it. This symptom needed no special interpretation, for Mr. — had come to me with some chronic syphilitic symptoms, slight in degree but of some standing, and for these a specific treatment was instituted. To our gratification, as the medicines influenced the system the cardiac troubles vanished, and the concomitant nervousness, which I had regarded as due to anxiety and overwork, fled away likewise. Of gastralgia, a very interesting example came but recently under my observation in the person of a young barrister, who had been infected with syphilis a year or two previously. He complained of 'atonic dyspepsia' and of nervous cravings at the stomach, which made him very uneasy. He had also very severe pains in the epigastric region, which tormented him sadly, and were not due to the ingestion of food, though he himself would call them indigestion. With all this he had in great measure the changed mental condition to which I have referred under my first heading. From being a very merry, light-hearted man, and full of gaiety and life, he had become querulous, irritable, so

despondent that he could scarcely detail his symptoms to me without bursting into tears, irresolute, too, and impatient, rushing off to one health resort and then another in the search for relief, and writing letters of desperation to me from each one of them. At his first visit I did not think of the syphilitic nature of the symptoms, which otherwise are not uncommon, and I ordered Easton's syrup. From this he gained relief, which, however, was but temporary; it was mere palliation, and his whole condition of unrest was but little the better; then I sent a prescription for arsenic to him, which answered no better. Then again at my suggestion he went for a yachting trip, and was, I thought, better, when he wrote to me from town a letter more anxious and disappointed than ever. I advised his seeing an eminent physician there with whom I corresponded, and we agreed that syphilis was at the root of the evil. He was treated accordingly, and spent a couple of months at Aix with excellent results. He writes from there that for the first time he now feels as if we had got to the root of the thing. All other remedies seemed to give but superficial relief; now he is conscious of a radical cure. I should not add to the strength of my remarks by relating more of those cases which the reader can supply from his own experience, and I would only say in conclusion that the space given me in these Reports may not have been unfruitfully given to me instead of to far abler contributors if obscure as well as obvious neuroses become better known as consequences of syphilis, and if thereby a few sufferers gain relief who under other remedies would have found no ease to their sufferings.

THE WEIGHT OF THE BRAIN

IN THE INSANE.

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The following statistics, which were originally compiled, in a more limited form, at the instigation of Professor Turner, are drawn from a consideration of 716 cases (404 males and 312 females), on which careful post-mortems were made. The whole subject matter is derived from the West Riding Asylum records of the last six years, and I am indebted to the courtesy of Dr. Crichton-Browne for the privilege of utilising it, as well as for assistance in its collection.

It will be seen on referring to the subjoined tables that I have considered the subject chiefly under three headings : (1) Age. (2) Disease (mental). (3) Nationality. I have further occasionally compared the brain weights occurring in the insane with those of sanity, and have shown how far the co-existence of a wasting bodily disease, complicating insanity, affects the weight of the brain.

In each case the maximum, minimum, and average weights of the brain (entire), and of the cerebellum, pons and medulla, taken collectively, are given, together with the average age. The tables are also divided, as regards sex, into male and female.

1. Age.

In estimating the average brain weight for age, all ages under 20 years are considered together, as also are all ages of 70 years and upwards. Ages intermediate to these are

arranged in decades, and the average struck off in each instance.

In forming the decades I have found it more convenient not to arrange the numbers in their natural order of 1 to 10, both inclusive, but rather to make 0 and 9 the terminal figures in each group. Thus, 20 forms the first figure to be considered in the decennial period ' 20 to 30,' and the 30 is not included; and so of the rest.

In eleven cases the age was unknown.

The total age of the 705 patients whose ages were ascertained amounted to 32,718 years, giving an average age of 46·408, &c., years per individual, the maximum age being 87 years and the minimum 10 years.

For males the total average age was 46·356, &c., years, and for females, 46·474, &c., years.

The total average brain weight for all ages and both sexes =46·285, &c., ozs.

For males alone, 48·149, &c., ozs., with a maximum brain weight of 61 ozs. and a minimum brain weight of 31 ozs.

For females alone, 43·872, &c., ozs., with a maximum brain of 56 ozs. and a minimum brain of 30 ozs.

In males the greatest average brain weight was attained between 50 and 60 years of age, and in females between 20 and 30 years of age.

The maximum male brain occurred in a case of senile dementia 70 years of age, and the minimum male brain in a case of dementia 72 years of age.

The maximum female brain occurred in a case of mania 33 years of age, and the minimum female brain in a case of senile dementia 67 years of age. One of the same weight also occurred in a case of epileptic dementia 40 years of age.

Quain, in his ' Elements of Anatomy,' [1] gives a table containing results obtained by Sims, Clendinning, Tiedmann, and J. Reid, from an examination of 278 male and 191 female brains, which fix the male maximum sane brain at 65 ozs., and the minimum at 34 ozs., whilst the female maximum sane brain is fixed at 56 ozs., and the minimum at 31 ozs.

[1] Vol. ii. p. 568.

Dr. Robert Boyd, on the other hand, gives,[1] from an examination of 2,086 brains of both sexes, a maximum male sane brain of 60¾ ozs., and a minimum of 30¼ ozs., and a female maximum sane brain of 55¼ ozs., with a minimum of 27½ ozs., which are all below those of insanity, as shown by tables under consideration. He also gives as the average brain weight for males above 14 years of age, 47·1 ozs., and for females above 14 years of age, 42·5 ozs.

These averages are below those drawn from the cases under consideration.

These facts would seem to illustrate the truth of Wagner's conclusion that superiority of size of brain cannot be regarded as a constant accompaniment of superiority of intellect.

Apropos of this subject I may state that the 9 male cases in which the age was unascertained, and who were criminals sent by the Secretary of State, had an average brain weight greater than the general average for the male sex, viz., 49·2 ozs., as compared with 48·149, &c., ozs.

The total average weight of the cerebellum, pons and medulla, for all ages and both sexes = 5·984, &c., ozs. For males alone it = 6·255, &c., ozs. For females alone = 5·626, &c., ozs.

Male maximum C. P. and M.[2] = 9 ozs., and occurred in a case of general paralysis 44 years of age. Female maximum C. P. and M. = 8 ozs., and occurred in a case of acute mania 42 years of age.

In males the greatest average C. P. and M. weight was attained between the ages of 30 and 40 years. In females the greatest average was attained between the ages of 40 and 50 years.

Dr. J. Reid, from an examination of 53 male and 34 female sane brains, between the ages of 25 and 45 years, gives the male average of the C. P. and M. as 6·234, &c., ozs., and the female average as 5·781, &c., ozs.

Huschke, from an examination of 38 male and 22 female sane brains, agrees with the above.

From this it appears that the C. P. and M. weigh heavier

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[1] 'Philosophical Transactions,' 1860.
[2] C. P. and M. is used for brevity, to signify Cerebellum, Pons, and Medulla.

in the insane than in the sane male, whilst the converse obtains in the case of the female.

The difference between the extreme weights of the encephalon in male lunatics = 30 ozs., and in female = 26 ozs.

The difference between the extreme weights of the C. P. and M. in male lunatics = 5 ozs., and in females it also = 5 ozs.

The total average for adult male brain = 48·239, &c., ozs., and for adult female brain = 43·910, &c., ozs.; for adult male C. P. and M. = 6·255, &c., ozs., and for female = 5·634, &c., ozs.

Under 20 *years of age.*—This group included 14 males and 9 females.

Analysis of the 14 male cases showed them to consist of: idiotcy, 4; imbecility, 1; dementia, 3; acute forms,[1] 1; epileptic insanity, 5.

Female cases (9): idiotcy, 3; dementia, 1; acute forms, 2; epileptics, 3.

Male maximum brain = $52\frac{1}{2}$ ozs.; minimum brain = 32 ozs.

Maximum C. P. and M. = 7 ozs.; minimum $5\frac{1}{2}$ ozs.; average = 6·25 ozs.

Female maximum brain = 48 ozs.; minimum = 34 ozs.

Maximum C. P. and M. = 7 ozs.; minimum = $4\frac{1}{2}$ ozs.; average = 5·428, &c., ozs.

Here the average brain weight (45·698, &c., ozs., for males, and 42·7 ozs. for females), is lowered by the idiots included in the group.

From 20 *to* 30 *years of age.*—Included 27 males, and 40 females.

Analysis of male cases: idiotcy, 1; dementia, 8; acute forms, 8; general paralysis, 2; epileptics, 8.

Female cases: imbecility, 2; dementia, 8; acute forms, 18; general paralysis, 1; epileptics, 11.

Male maximum brain = $58\frac{1}{2}$ ozs.; minimum = 34 ozs.; and average = 48·5370 ozs.

Maximum C. P. and M. = $7\frac{1}{2}$ ozs.; minimum = 5 ozs.; average = 6·104, &c., ozs.

Female maximum brain = 54 ozs.; minimum = 34 ozs.; average = 44·681, &c., ozs.

[1] This expression (acute forms) is used as an abbreviation of the class styled in the Tables 'Mania, Melancholia and Acute Forms of Insanity.'

Maximum C. P. and M.=7 ozs.; minimum=$4\frac{3}{4}$ ozs.; and average=5·742, &c., ozs.

Here the brain averages for both sexes are large, from the number of acute forms included, and in this decade the female brain reaches its largest average.

From 30 *to* 40 *years of age.*—Included 90 males, and 65 females.

Analysis of male cases: idiotcy, 2; dementia, 14; acute forms, 18; general paralysis, 40; epileptics, 10; chronic mania, 6.

Females: imbecility, 1; dementia, 8; acute forms, 31; general paralysis, 11; epileptics, 8; chronic mania, 6.

Male maximum brain=58 ozs.; minimum=$33\frac{1}{4}$ ozs.; average=48·0305 ozs.

Maximum C. P. and M.=8 ozs.; minimum=$4\frac{1}{2}$ ozs.; average=6·444, &c., ozs.

Female maximum brain=56 ozs.; minimum=34 ozs.; average=44·315, &c., ozs.

Maximum C. P. and M.=7 ozs.; minimum=$4\frac{1}{2}$ ozs.; average=5·627, &c., ozs.

From 40 *to* 50 *years of age.*—Included 109 male, and 66 females.

Analysis of males: dementia, 30; acute forms, 18; general paralysis, 53; epileptics, 4; chronic mania, 4.

Females: dementia, 16; acute forms, 24; general paralysis, 11; epileptics, 7; chronic mania, 8.

Male maximum brain=$60\frac{1}{2}$ ozs.; minimum=37 ozs.; average=48·323, &c., ozs.

Maximum C. P. and M.=9 ozs.; minimum=$4\frac{1}{2}$ ozs.; average=6·294 ozs.

Female maximum brain=53 ozs.; minimum=30 ozs.; and average=44·382, &c., ozs.

Maximum C. P. and M.=8 ozs.; minimum=4 ozs.; and average=5·745 ozs. Largest male C. P. and M. occurs in this decade, as also does the largest female C. P. and M.

From 50 *to* 60 *years of age.*—Included 80 males, and 59 females.

Analysis. Males: imbecility, 1; dementia, 44; acute

forms, 16; general paralysis, 13; epileptics, 1; chronic mania, 5.

Females: dementia, 28; senile dementia, 2; acute forms, 15; general paralysis, 2; epileptics, 2; chronic mania, 10.

Male maximum brain = 59½ ozs.; minimum = 40 ozs.; and average = 48·875 ozs.

This is the highest brain average reached in the decennial periods.

Maximum C. P. and M. = 7½ ozs.; minimum = 4 ozs.; average = 6·227 ozs.

Female maximum brain = 55 ozs.; minimum = 35 ozs.; and average = 43·444, &c., ozs.

Maximum C. P. and M. = 6½ ozs.; minimum = 4½ ozs.; average = 5·651, &c., ozs.

From 60 *to* 70 *years of age.*—Included 57 males, and 44 females.

Analysis. Males: dementia, 24; senile dementia, 18; acute forms, 8; general paralysis, 2; epileptics, 2; chronic mania, 3.

Females: dementia, 13; senile dementia, 21; acute forms, 4; epileptic, 2; chronic mania, 4.

Male maximum brain = 58 ozs.; minimum = 35 ozs.; and average = 47·640, &c., ozs.

Maximum C. P. and M. = 8 ozs.; minimum = 4½ ozs.; average = 6·158, &c., ozs.

Female maximum brain = 50 ozs.; minimum = 30 ozs.; and average = 43·636 ozs.

Maximum C. P. and M. = 6½ ozs.; minimum = 4½ ozs.; average = 5·5625 ozs.

70 *years of age and upwards.*—Included 18 males, and 27 females.

Analysis of cases. Males: dementia, 3; senile dementia, 15.

Females: dementia, 1; senile dementia, 22; acute forms, 2; epileptic, 1; chronic mania, 1.

Male maximum brain = 61 ozs.; minimum = 31 ozs.; extreme difference = 30 ozs.; average = 46·861 ozs.

Maximum C. P. and M. = 7½ ozs.; minimum = 5 ozs.; average = 5·859, &c., ozs.

Female maximum brain=50 ozs. ; minimum=36 ozs. ; average=42·3 ozs.

Maximum C. P. and M.=7 ozs.; minimum=3 ozs.; average=5·2083 ozs.

This section included the largest and the smallest male brains, and the smallest female C. P. and M., in the tables.

Age unknown.—Included 9 males and 2 females.

Analysis of cases. Males: dementia, 2 ; acute forms, 1 ; general paralysis, 3 ; epileptic, 1 ; chronic mania, 2.

Females : acute forms, 1 ; senile dementia, 1.

Male maximum brain=54 ozs. ; minimum=43 ozs. ; and average=49·2 ozs.

Maximum C. P. and M.=7 ozs.; minimum=5 ozs.; average=6·3125 ozs.

Female brains weighed 42 and 38 ozs., and the C. P. and M. $7\frac{1}{2}$ and $5\frac{1}{2}$ ozs. respectively, giving corresponding averages of 40·0 ozs. and 6·5 ozs.

2. Disease.

Considerable fluctuations in the weight of the brain occurred in the several varieties of insanity. The acute forms showed the highest average and idiotcy the lowest.

The maximum male brain occurred in senile dementia, and the minimum in dementia.

The maximum female brain occurred in acute forms, and minimum in epileptic insanity.

Maximum male C. P. and M. occurred in general paralysis, and the minimum in the acute forms.

Female maximum C. P. and M. occurred in the acute forms, and the minimum in senile dementia.

Idiotcy showed the lowest average C. P. and M. in both sexes, whilst the highest average was shown in imbecility for males and in chronic mania for females.

An analysis of the 716 cases included in the tables shows, *the males* (404) to consist of—idiotcy, 7 ; imbecility, 2 ; dementia, 128 ; senile dementia, 33 ; acute forms, 70 ; general

paralysis, 113 ; epileptic insanity, 31 ; chronic mania, 20 ; *and the females* (312) to consist of—idiotcy, 3 ; imbecility, 3 ; dementia, 75 ; senile dementia, 46 ; acute forms, 97 ; general paralysis, 25 ; epileptic insanity, 34 ; and chronic mania, 29.

Idiotcy.—Only 10 cases[1] occurred in tables.

Male maximum brain = 54 ozs., and the minimum = 34 ozs. ; average = 41·928, &c. ozs. Maximum age reached = 39 years, and minimum = 10 years, with an average age of 21·857, &c. years.

The brains here were large as compared with three mentioned by Tiedemann, whose average brain weight = 22·6 ozs. for an average age of 35·3 years.

Male maximum C. P. and M. = 6 ozs.; minimum = 5½ ozs.; average = 5·83 ozs.

The female brains = 36, 34 and 42 ozs. respectively, giving an average of 37·3 ozs. for an average age of 17 years.

Sims mentions a female idiot, 12 years of age, whose brain only weighed 27 ozs.

Only one female C. P. and M. was weighed = 5 ozs.

Extreme difference : brain = 20 ozs. ; C. P. and M. = 1 oz.

Imbecility.—Comprised 2 males and 3 females.

Male brains weighed 52 and 50 ozs. and the C. P. and M. 7 and 6½ ozs., giving an average respectively of 51 ozs. and 6·75 ozs. for an average age of 35 years.

Female brains weighed 44, 41, and 50 ozs., and one C. P. and M. which weighed 5½ ozs.

Average brain = 45·0 ozs., for an average age of 29·6 years.

Extreme difference : brain = 11 ozs. ; C. P. and M. = 1½ oz.

This section consists of too limited a number of cases to furnish any useful results.

Dementia. — 203 cases examined : 128 males and 75 females.

Male maximum brain = 60½ ozs. ; minimum = 31 ozs. ; and average = 48·0117, &c. ozs., for an average age of 49·8809, &c. years.

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[1] Epileptic idiots are not included in this class, but come under the head of Epileptic Insanity.

C. P. and M. maximum = 8 ozs.; minimum = 4½ ozs.; average = 6·224, &c. ozs.

Female maximum brain = 51½ ozs.; minimum = 35 ozs.; and average = 43·773 ozs., with an average age of 48·346 years.

C. P. and M. maximum = 6¾ ozs.; minimum = 4 ozs.; average = 5·597, &c. ozs.

Extreme difference: brain = 29½ ozs.; C. P. and M. = 4 ozs.

Senile Dementia.—79 cases examined: 33 males and 46 females.

Male maximum brain = 61 ozs.; minimum = 38¾ ozs.; and average = 47·477, &c. ozs., for an average age of 69·36 years.

C. P. and M. maximum = 7½ ozs.; minimum = 5 ozs.; average = 5·990, &c. ozs.

Female maximum brain = 50 ozs.; minimum = 30 ozs.; and average = 42·510, &c. ozs., for an average age of 69·4 years.

C. P. and M. maximum = 7 ozs.; minimum = 3 ozs.; average = 5·307, &c. ozs.

Extreme difference: brain = 31 ozs.; C. P. and M. = 4½ ozs.

Senile dementia furnishes the largest male and the smallest female brains, and the smallest female C. P. and M.

Mania, Melancholia, and Acute forms of Insanity.—This division shows the largest average brains, both for males and females, but the superiority does not extend to the C. P. and M.

167 cases examined: 70 males and 97 females.

Male maximum brain = 60 ozs.; minimum = 42 ozs.; and average = 51·0214, &c. ozs., for an average age of 43·7101, &c. years.

C. P. and M. maximum = 8 ozs.; minimum = 4 ozs.; average = 6·469, &c. ozs.

Female maximum brain = 56 ozs.; minimum = 36 ozs.; and average = 45·494, &c. ozs., for an average age of 40·062, &c. years.

C. P. and M. maximum = 8 ozs.; minimum = 4 ozs.; average = 5·806 ozs.

Extreme difference : brain=24 ozs.; C. P. and M.=4 ozs. The largest female brain is included in this division.

General Paralysis.—138 cases examined : 113 males and 25 females.

Male maximum brain=58 ozs.; minimum=$33\frac{1}{4}$ ozs.; and average=46·522, &c. ozs., for an average age of 42·045 years.

C. P. and M. maximum=9 ozs.; minimum=$4\frac{1}{2}$ ozs.; average=6·230, &c. ozs.

Female maximum brain=48 ozs.; minimum=32 ozs.; and average=40·73 ozs., for an average age of 39·92 years.

C. P. and M. maximum=7 ozs.; minimum=$4\frac{1}{2}$ ozs.; average=5·5119, &c. ozs.

Extreme difference : brain=26 ozs.; C. P. and M.=$4\frac{1}{2}$ ozs. This disease includes the largest male C. P. and M.

Epileptic Insanity.—65 cases examined : 31 males and 34 females.

Male maximum brain=56 ozs.; minimum=40 ozs.; and average=49·225, &c. ozs., for an average age of 32·86 years.

C. P. and M. maximum=8 ozs.; minimum=5 ozs.; average =6·261, &c. ozs.

Female maximum brain=$50\frac{1}{2}$ ozs.; minimum=30 ozs.; and average=43·022, &c. ozs., for an average age of 36·147, &c. years.

C.P. and M. maximum=7 ozs.; minimum=$4\frac{1}{2}$ ozs.; average=5·525, &c. ozs.

Extreme difference : brain=26 ozs.; C. P. and M.=$3\frac{2}{3}$ ozs.

Chronic Mania.—49 cases examined : 20 males and 29 females.

Male maximum brain=$58\frac{1}{2}$ ozs.; minimum=41 ozs.; and average=49·5 ozs., for an average age of 48·16 years.

C. P. and M. maximum=$7\frac{1}{2}$ ozs.; minimum=$5\frac{1}{2}$ ozs.; average=6·27 ozs.

Female maximum brain=55 ozs.; minimum=37 ozs.; and average=45·129, &c. ozs., for an average age of 49·827, &c. years.

C. P. and M. maximum=7 ozs.; minimum—5 ozs.; average=5·906, &c. ozs.

Extreme difference : brain=$21\frac{1}{2}$ ozs.; C. P. and M.= $2\frac{1}{2}$ ozs.

Chronic mania has the largest female C. P. and M. average.

Insanity complicated by Phthisis.—The co-existence of a wasting bodily disease does not appear to lower the average brain weight. Thus, 146 of the 716 cases examined were complicated with phthisis, 62 of them being males and 84 females.

Of these, the 62 males included—idiotcy, 3 ; imbecility, 1 ; dementia, 24 ; acute forms, 16 ; general paralysis, 6 ; epileptics, 7 ; chronic mania, 5.

The 84 females included—idiotcy, 2 ; imbecility, 3 ; dementia, 19 ; senile dementia, 9 ; acute forms, 30 ; general paralysis, 1 ; epileptics, 8 ; chronic mania, 12.

Males had a maximum brain of $58\frac{1}{2}$ ozs., a minimum brain of 31 ozs., and an average brain of 48·197, &c. ozs., for an average age of 39·885 &c. years.

C. P. and M. maximum $= 7\frac{1}{2}$ ozs. ; minimum $= 4\frac{1}{2}$ ozs. ; average $= 6·22$ ozs.

Females had a maximum brain of 55 ozs., a minimum brain of 34 ozs., and an average brain of 44·086, &c. ozs., for an average age of 41·506, &c. years.

C. P. and M. maximum $= 6\frac{1}{2}$ ozs. ; minimum $= 4\frac{1}{2}$ ozs. ; average $= 5·612$, &c. ozs.

Extreme difference : brain $= 27\frac{1}{2}$ ozs. ; C. P. and M. $= 3$ ozs.

In the case of both males and females, therefore, the brain was above the general average, whilst the C. P. and M. was below it.

From a consideration of the individuals forming this group it would seem that two classes of lunatics are peculiarly obnoxious to phthisis—viz. the demented, who can not, and those labouring under the acute forms, who will not, help themselves or complain. But the development of this and other theories involved in these tables I leave to abler and more experienced hands, being content to act as a faithful recorder of facts observed.

3. Nationality.

The great majority of the cases were, from the situation of the Asylum, necessarily English, this class numbering 363

males and 279 females; but the proportion of Irish included (26 males and 29 females) allows of a very fair average being deduced for that nation. As for the Scottish and foreign averages, they must be taken for what they are worth: at least their elimination renders more clear the value of the rest.

English.—642 cases examined : 363 males and 279 females.

Male maximum brain = 61 ozs.; minimum = 31 ozs.; and average = 48·15909 ozs., for an average age of 46·435, &c. years.

C. P. and M. maximum = 9 ozs.; minimum = 4 ozs.; average = 6·253, &c. ozs.

Female maximum brain = 56 ozs.; minimum = 30 ozs.; and average = 43·803, &c. ozs., for an average age of 46·924, &c. years.

C. P. and M. maximum = 8 ozs.; minimum = 3 ozs.; average = 5·610, &c. ozs.

Extreme difference : brain = 31 ozs.; C. P. and M. = 6 ozs.

Irish.—55 cases examined: 26 males and 29 females.

Male maximum brain = 55 ozs.; minimum = 38 ozs.; and average = 48·134, &c. ozs., for an average age of 47·391, &c. years.

C. P. and M. maximum = 7 ozs.; minimum = 5½ ozs.; average = 6·190, &c. ozs.

Female maximum brain = 55 ozs.; minimum = 36 ozs.; and average = 44·310, &c. ozs., for an average age of 42·655, &c. years.

C. P. and M. maximum = 7 ozs.; minimum = 4½ ozs.; average = 5·738, &c. ozs.

Extreme difference : brain = 19 ozs.; C. P. and M. = 2½ ozs.

Scottish.—Male brains, of which there were only 3, weighed respectively 51, 49 and 44 ozs., giving an average of 48·0 ozs. for an average age of 55·6 years.

C. P. and M. were also 3 in number, and weighed respectively 6½, 6 and 6 ozs., giving an average of 6·16 ozs.

There were no females.

Foreign.—5 cases examined : 4 males and 1 female.

Male maximum brain = 50½ ozs.; minimum = 37 ozs.; and average = 45·375 ozs., for an average age of 39·25 years.

C. P. and M. maximum = 6¼ ozs.; minimum = 5½ ozs.; average = 5·916 ozs.

Only one female, who was 32 years of age, and whose brain = 42 ozs. and C. P. and M. = $5\frac{1}{2}$ ozs.

Extreme difference : brain = $13\frac{1}{2}$ ozs.; C. P. and M. = $\frac{3}{4}$ oz.

Nationality unknown.—11 cases examined : 8 males and 3 females.

Male maximum brain = 56 ozs.; minimum = 42 ozs.; and average = 49·187, &c. ozs., for an average age of 39 years.

C. P. and M. maximum = $7\frac{1}{2}$ ozs.; minimum = 6 ozs.; average = 6·714, &c. ozs.

The 3 female brains weighed respectively 48, 50 and 42 ozs., giving an average of 46·6 ozs. for an average age of 46·6 years.

C. P. and M. = respectively 6, $6\frac{1}{2}$ and $5\frac{1}{2}$ ozs.; average = 6 ozs.

Extreme difference : brain = 14 ozs.; C. P. and M. = 2 ozs.

The averages for this group are very high indeed, being raised by the dementia and acute forms.

The 8 males consisted of—dementia, 1 ; acute forms, 3 ; general paralysis, 4.

The 3 females of—dementia, 3.

The subjoined is an abstract of the more elaborate tables in my possession, and shows at a glance the principal points treated of, more at length, in the foregoing paper.

ABSTRACT FROM TABLES ON BRAIN WEIGHT.

Average Weight of Brain (entire) and of Cerebellum, Pons, and Medulla in relation to—

	ALL CASES			MALE			FEMALE		
	Brain	C. P. and M.	Average Age	Brain	C. P. and M.	Average Age	Brain	C. P. and M.	Average Age
1. *Age.*									
Under 20 years	44·554, &c.	5·890, &c.	17·565, &c.	45·698, &c.	6·25	17·214, &c.	42·7	5·428, &c.	18·1
20 to 30	46·235, &c.	5·892, &c.	25·462, &c.	48·5370	6·104, &c.	25·703	44·681, &c.	5·742, &c.	25·3
30 „ 40	46·472, &c.	6·107, &c.	34·974, &c.	48·0305	6·444, &c.	35·26	44·315, &c.	5·627, &c.	34·569, &c.
40 „ 50	46·837, &c.	6·086, &c.	44·468, &c.	48·323, &c.	6·294	44·706, &c.	44·382, &c.	5·745	44·075
50 „ 60	46·570, &c.	5·984, &c.	54·323, &c.	48·875	6·227	54·1625	43·444, &c.	5·651, &c.	54·542, &c.
60 „ 70	45·896, &c.	5·922, &c.	64·207, &c.	47·640, &c.	6·158, &c.	63·824, &c.	43·636	5·5625	64·7045
70 years and upwards	44·14	5·468, &c.	74·84	46·861	5·859, &c.	75·4	42·3	5·2083	74·481
Age unknown	47·54	6·35	...	49·2	6·3125	...	40·0	6·5	...
All ages	46·285, &c.	5·984, &c.	46·408, &c.	48·149, &c.	6·255, &c.	46·356, &c.	43·872, &c.	5·626, &c.	46·471, &c.
2. *Disease.*									
Idiotcy	40·55	5·625	20·4	41·928, &c.	5·83	21·857, &c.	37·3	5·0	17·0
Imbecility	47·4	6·3	31·8	51·0	6·75	35·0	45·0	5·5	29·6
Dementia	46·445, &c.	6·0029, &c.	49·358, &c.	48·0117, &c.	6·224, &c.	49·8809, &c.	43·773	5·597, &c.	48·346
Senile Dementia	44·585, &c.	5·587, &c.	69·384, &c.	47·477, &c.	5·990, &c.	69·36	42·510, &c.	5·307, &c.	69·4
Mania, Melancholia, and Acute forms of Insanity	47·811, &c.	6·095, &c.	41·587, &c.	51·0214, &c.	6·469, &c.	43·710, &c.	45·494, &c.	5·806	40·062, &c.
General Paralysis	45·472, &c.	6·095, &c.	41·725, &c.	46·522, &c.	6·230, &c.	42·045	40·73	5·5119, &c.	39·92
Epileptic Insanity	45·980, &c.	5·843, &c.	34·609, &c.	49·225, &c.	6·261, &c.	32·86	43·022, &c.	5·525, &c.	36·147, &c.
Chronic Mania	46·913, &c.	6·065, &c.	49·191, &c.	49·5	6·27	48·16	45·129, &c.	5·906, &c.	49·827, &c.
3. *Nationality.*									
English	46·266, &c.	5·976, &c.	16·648, &c.	48·15909	6·253, &c.	46·435, &c.	43·803, &c.	5·610, &c.	46·924, &c.
Scottish	...	...	...	48·0	6·16	55·5	...	...	...
Irish	46·118	5·959, &c.	44·75	48·134, &c.	6·190, &c.	47·391, &c.	44·310, &c.	5·738, &c.	42·655, &c.
Foreign	44·7	5·8125	37·8	45·375	5·916	39·25	42·0	5·5	32·0
Unknown	48·5	6·5	41·3	49·187, &c.	6·714, &c.	39·0	46·6	6·0	46·6

The above weights are expressed in ounces and decimals of ounces, and the ages in years and decimals of years.

CHANGE OF LIFE, AND INSANITY.

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THERE are certain periods in the lives of all women which are especially liable to attacks of nervous disorder. The most perilous of these epochs corresponds with those conditions of active uterine function included in the phenomena of pregnancy, parturition, and lactation. But next to these, in susceptibility, must be ranked the ages marked by the advent and the departure of the catamenial discharge.

The following observations apply to one of the morbid conditions connected with the termination of uterine function, namely, insanity occurring at the change of life.

It is an acknowledged fact that, even in healthy women, the climacteric is not usually encountered without some slight derangement of the mental and bodily equilibrium. But, beyond the nervous disturbance which might naturally be expected to occur, it is probable that cerebral disorder amounting to actual insanity is extremely rare at the change of life.[1]

According to the statistics of Dr. Tilt, amongst 500 women affected with nervous diseases at the ménopause, 16 only became insane and 3 epileptic.

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[1] 'Diseases occurring at are not the same as diseases caused by the change of life.'—' Tilt on the Change of Life,' p. 75.

But as in hospitals and asylums we are thrown into contact with exceptional conditions of health, it is there we have an opportunity to enquire if the morbid conditions associated with the change of life have any set of mental symptoms peculiar to themselves, which separate and distinguish them from cases of insanity occurring at other ages.

With this object an investigation has been made concerning the cases of 100 women, who were admitted to the West Riding Asylum during the last thirteen years, in whom an attack of insanity could be distinctly traced as occurring at, although not necessarily caused by, the change of life.

An enquiry into the history and symptoms of these cases has led to the following conclusions :

(1). That insanity occurring at the change of life is not usually caused by that condition *per se,* but is most frequently due to some other moral or physical cause coincident with that critical period.

(2). That the age most liable to an attack of insanity at the climacteric is 45 years and 2 months.

(3). That the onset of the cerebral disorder generally occurs at an interval of one year from the date at which the catamenial discharge ceases to occur.

(4). That the married state does not appear to predispose a woman to mental aberration at the climacteric more than a condition of celibacy.

(5). That the number of children which a woman gives birth to does not appear to bear any relation to her liability to cerebral disorder at the occurrence of the ménopause.

(6). That the forms of insanity most common at the change of life are melancholia, and, more rarely, mania ; and that dementia, epileptic insanity, and general paralysis are very unusual forms as occurring originally at the climacteric, the forms on admission being as follows—melancholia, 67 ; mania, 24 ; dementia, 4 ; epileptic insanity, 3 ; and general paralysis, 2.

(7). That there is a certain group of symptoms manifested in insanity at the change of life which, if taken together,

would enable a person to diagnose a case of mental aberration connected with that period, independently of a knowledge of the history of the case or of the age of the patient.

(8). That the prognosis in insanity occurring at the change of life is decidedly favourable, the recoveries being 40 per cent. of those attacked.

(9). That the duration of the disease is usually more than three months and less than three years, and that complete recovery is not to be expected until twelve months after the commencement of the attack.

(10). That, with regard to treatment, mild sedatives and aperients, a careful watchfulness for suicidal tendencies, and the observance of a quiet and regular course of life, are chiefly indicated.

Causation.

An enquiry into the history of the patients has led to the conclusion that the change of life *per se* is not frequently a cause of mental disorder. It is when some great moral shock is added to the agencies already disturbing the nervous system of women at this period, that we may look for an attack of insanity.

In 40 cases some such exciting cause could be distinctly traced. In the remaining cases there was either some doubt as to the causation of the attack, or no satisfactory history could be obtained. In these 40 instances, 28 cases were apparently due to moral and 12 to physical causes.

The moral causes were as follows :—Deaths in the family in 10 cases ; cruelty or infidelity of the husband in 7 ; religious excitement in 4 ; pecuniary anxiety in 3 ; fright in 3 ; and disappointment in love in 1 case.

The physical causes were—fever in 2 cases ; overwork in 2 ; intemperance in 2 ; prostitution in 2 ;[1] inflammation of the uterus in 1 ; menorrhagia in 1 ; fall on the head in 1 ; and excessive smoking in 1 case each.

[1] 'Les femmes qui vivent dans le libertinage franchissent difficilement la ménopausie.'—Gardanne, ' *Avis aux femmes qui entrent l'âge critique,*' p. 234.

Many cases were preceded by hysterical or hypochondriacal symptoms, and almost all the patients had been previously somewhat out of health. In 2 cases there was a history of severe piles, in another of successive crops of boils, and in another of an ovarian tumour.

In 19 cases there was a distinct hereditary taint recorded, but the proportion was probably much larger. Thirty-six had had previous attacks, which were in many instances associated with the puerperal condition.

The following interesting questions presented themselves for solution in this enquiry, to which the case-books, for the most part, returned satisfactory answers :—

First. What age is most susceptible to cerebral disorder connected with the ménopause ?

The age most liable to an attack of insanity at the change of life is 45 years and 2 months, according to the following calculations : Taking 45 years as the age at which the attack usually occurs, 43 and 44 being the two years previous to that age, and 46 and 47 the two years subsequent to it, we find that in these five years no less than 39 women out of the 100 became insane. In the previous five years—namely, at the ages 38, 39, 40, 41 and 42—only 21 became insane. During the five years subsequent to the age 47—namely, at the ages 48, 49, 50, 51 and 52—only 28 became insane.

In the five years preceding the age 38—namely, at the ages 33, 34, 35, 36 and 37—only 6 became insane. In the five years following the age 52—namely, at the ages 53, 54, 55, 56 and 57—only 3 became insane.

One woman became insane at the age of 31 and another at the age of 58 from causes associated with the change of life, but these were exceptional cases.

Second. What is the interval of time between the cessation of the catamenial discharge and the attack ?

It would appear that an attack of insanity is not to be looked for either at the time the discharge ceases altogether, nor even during the first few months subsequent to cessation. Two premises are necessary to arrive at any approximate conclusion on this point. The first is, the exact age at which

the catamenia ceased to appear; and the second is, the exact date at which the morbid mental symptoms were first noticed.

In only 41 cases out of the 100 could even a rough estimate be formed on these two points. But, allowing for the usual inaccuracies of relatives in giving the history of the case, and bringing to bear the course of events as they usually occur in healthy women, the following statements may, it is believed, be considered fairly trustworthy:

In 2 cases the attack occurred two months after cessation; in 3 cases, three months after it; in 3 cases, after an interval of five months; in 2 cases, after six months; in 1 case, after seven months; in 3 cases, after nine months; in 1 case, after ten months; in 1 case, after eleven months; in no less than 13 cases, after twelve months; in 1 case, after fourteen months; in 6 cases, after twenty-four months; in 1 case, after thirty-six months; and in 3 cases, after forty-eight months.

It appears, therefore, that the period most liable to an attack of insanity is about one year after the catamenial discharge has ceased to occur.

Thirdly. Are married or single women more liable to an attack of insanity at the change of life?

The married state does not appear either to predispose a woman to, or to exempt her from, an attack at the méno-pause.

Amongst 99 women whose condition as to marriage was recorded, 72 were married (including 7 widows) and 27 were single. This proportion of married women to single is not very different to that which is found to exist amongst the sane and healthy.

The cause of the insanity in the two cases is, however, somewhat different.

In the case of the married woman, there is the nervous disturbance which occurs at the change of life added to the effects of the shocks which have been sustained during previous pregnancies. 'Thus many women may have passed through the trials of puberty and child-bearing, and will

break down at the ménopause. Often, no doubt, this is the climax—the last ounce of a long-troubled sexual life.'[1]

But, in the case of the single woman, the occurrence of insanity at the climacteric has a different signification. Here it may be regarded as the crowning trouble of a life of sexual disappointment ; and, in addition to the presence of nervous uterine disturbance, we have to allow for the moral cause which is probably at work, which has been appropriately named by the French *amour propre blessé*.

In both cases, however, as before observed, the cessation of the catamenial discharge is usually a mere predisposing cause, the immediately exciting cause of the attack being, in most instances, traceable to some severe physical or mental shock occurring at the climacteric.

Fourth. Do large or small families predispose the mother to an attack of insanity at the change of life ?

The number of children that a woman bears does not seem to render her more liable to an attack of insanity at the change of life, whatever other affections may result from having large families.

The number of children which the patients had had was accurately ascertained in 36 cases.

In 6 instances the woman had given birth to 1 child; in 7 to 2 children ; in 6 to 3 ; in 2 to 4 ; in 4 to 5 ; in 3 to 6 ; in 4 to 7 ; in only 1 to 8 ; in only 1 to 10 ; and in 2 to 11.

The greatest number of cases, therefore, occurred after the birth of 2 children ; and the average number of children which a woman has after which an attack of insanity may be expected, is 5.

The number of children, therefore, which a woman gives birth to does not appear to bear any relation to her liability to cerebral disorder at the occurrence of the ménopause.

BODILY CONDITIONS.

Most of the women were suffering from debility in some form or another at the time of the attack, and a large

[1] 'Lumleian Lectures,' by Robert Barnes, M.D., April 1873.

majority presented an anæmic, woe-begone appearance. The skin was usually dry and hot, and although the face was pale, there were the occasional hot flushes, together with cold sweatings of the extremities, so common at the change of life. A very large proportion of the women had dark hair and complexions, the exceptions to these appearances being very rare. The skin in many cases was yellow and jaundiced, and with this there was frequently present a condition of extreme nervous irritability. These combinations confirm the opinion of Dr. Tilt, that 'women are more liable to insanity' at the change of life, 'if the nervous be associated with the bilious temperament.' Dyspepsia, accompanied with loss of appetite and a foul tongue, was common, and the bowels were almost always constipated on admission. The respiration was frequently oppressed in the manner peculiar to chlorosis, the pulse was slow, feeble, and compressible. The urine was usually abundant and limpid, but in exceptional cases was scanty and loaded with lithates. Some patients, under a delusion, refused to pass water for some hours or even days.

The menses were either very scanty at the time of the attack, or had disappeared some months previous to it. In one or two cases leucorrhœa existed, but this was not commonly observed.

FORM OF INSANITY.

The attack usually commenced with symptoms of melancholia, which in many cases passed into mania. Delusions were common, but hallucinations were not frequently met with. General paralysis, occurring at the change of life, was only met with in 2 instances, epileptic insanity in 3, dementia in 4, and hemiplegia in 5 cases.

MENTAL SYMPTOMS.

In addition to the usual premonitory indications of insanity, such as headache, loss of sleep, and change of character, being noticed, there were a few symptoms especially

worthy of remark.   Perhaps the first and most constant
peculiarity observed was a neglect of household duties, or a
performance of them in some such irregular manner as to
attract the attention of the patient's relations.   One woman
proceeded to sweep the street clean in front of her house for
a long distance round it; another would carry pots and
pans to and fro, and be incessantly busy without really
getting on with her domestic duties.   A third would spend
the whole day in moving the furniture about from room to
room in a purposeless manner.   Almost all the patients were
restless at first, and slept badly.   Many wandered about the
house at night undressed.   Others strayed away from home,
often insufficiently clothed, and were taken up by the police,
being unable to give a satisfactory account of themselves.
Depression was frequently noticed, being sometimes so
marked that the patient would sit all day with her head in
her hands and refuse to speak.   With all these symptoms
there was a decided loss of mental tone, the memory became
impaired, and the conversation vague and incoherent.   The
most prominent symptom of all, however, was the extreme
restlessness exhibited by nearly all the patients in the early
stages of the attack.

This perturbation was usually succeeded by a condition of
extreme despondency, hysterical symptoms being occasionally
developed at this period.   Next, vague fears of some unde-
finable evil, sometimes connected with religious doubts,
were complained of.   The patient would then probably hint
that she was not good enough to live, and this notion would
gradually ripen till she took some actual measures to destroy
herself.   These impulses were not usually acted upon in a
sudden or unexpected manner, but the steps towards the
attempt were generally taken so gradually as to give the
relations plenty of time to prevent anything serious occurring.

Up to this point the patient, although suicidal, was not
usually obnoxious to others ; but soon a new phase of the
disease was displayed.   This symptom consisted of ground-
less suspicions of relatives and others, whom she fancied
were concocting all kinds of plots against her.   These
suspicions were constantly dwelt upon, and if the patient did

not make some attempt upon her own life which necessitated her removal to an asylum, she probably soon became violent and abusive, although absolutely homicidal tendencies were but rarely developed.

Others, again, became destructive, breaking furniture and glass if thwarted in any way. Soon another stage of the disease appeared, and the patient either laboured under some delusion, or became demented and incoherent for the time being. Hallucinations were also occasionally manifested, but their occurrence was rare. Swearing and obscenity of language were also uncommon, but when these symptoms did exist they were displayed to an extraordinary degree.[1]

Now all these symptoms, it may be said, are not unlike those exhibited in an ordinary case of melancholia or mania; and it is not intended in this paper to affirm that any one symptom, taken by itself, is pathognomonic of insanity at the change of life. All that is maintained is, that there is a certain group of symptoms manifested in the mental aberration occurring at that epoch which, if taken together, would enable a person to diagnose a case of cerebral disorder connected with the climacteric, independently of a knowledge of the history of the case or of the age of the patient.

Amongst the premonitory symptoms, neglect of household work was observed in 63 cases, restlessness and wandering about the house in 40 cases, straying away from home without an object in 16 cases, and stripping off clothes in 5 cases.

Amongst the more advanced symptoms were noticed extreme depression, amounting to melancholia, in no less than 67 cases, suicidal tendencies in 56 cases, refusal of food in 21 cases, religious anxiety in 20 cases, and extreme fear in 7 cases.

As the disease progressed, suspicions of relatives and others were noticed in 23 cases, great excitement in 30 cases,

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[1] 'J'ai pareillement observé que des tendences érotiques se montrent dans ces circonstances avec une intensité désespérante.'—Morel, *Traité des Maladies mentales*, p. 196.

violence to others in 14 cases, destruction of clothes and furniture in 19 cases, and threats in 39 cases.

In cases where dementia was the form of insanity, there was incoherence in 40 cases, and loss of memory in 18 cases.

Delusions were observed in 50 cases, and hallucinations in 13 cases.

In 5 cases, swearing and blasphemy were prominent symptoms, and in 3 cases the language became very obscene and erotic.

Some of the above symptoms are worthy of being considered in detail.

Extreme depression.—This was manifest in different ways, some passive, others more active. Amongst these melancholic cases, it was reported that one woman had remained silent for six months, others had become indolent and lethargic about their household duties, others neglected to eat or refused food, others were oppressed with religious doubts and fears, and a large proportion became suicidal in various degrees.

The suicidal tendencies exhibited by these patients may be roughly divided into four groups :

1st. One in which the patients confessed, after some pressure, that they had contemplated suicide.

2nd. One in which the patients, without any pressure, but of their own accord, stated that they intended to commit suicide.

3rd. One in which they took some decided steps towards committing the act, as by buying poison or by concealing weapons.

And 4th. One in which some actual attempt was made, as by swallowing laudanum or cutting the throat.

Fifty-six patients were returned as suicidal. In almost all the cases, the tendency manifested itself so gradually and yet so openly, that if any real attempt was made, the relatives were decidedly to blame for not taking steps to prevent such an occurrence.

Specimens of each of the above four stages were noted down as being of some interest.

In the first stage, one patient confessed, after much pressure, that she had contemplated suicide ; and others said they frequently felt tempted to destroy themselves, but took no more steps in the matter. Some of these cases, however, by neglect got on gradually to the more dangerous stages.

In the second stage, one woman refused all nourishment, and said that she and all her family must die together ; another affirmed that her soul was lost and she must destroy herself ; others said that théy wished to die, as they had no right to live, and were determined on self-destruction. Others specified the method they would prefer should be made use of. Several would have liked to be hanged, others to die of starvation, one threatened to poison herself, and another said that if she could get a knife she would cut her head off with it.

In the third stage, where some steps had been actually taken, one woman had concealed a rope and a razor with intent ; another was found in bed with a carving-knife ; a third attempted to procure a razor, and also to jump into a mill-pond ; and a fourth stated that she had often attempted to destroy herself by holding her breath. If the patient got as far as this third stage, and was not speedily interfered with, something more disastrous usually took place.

In the fourth stage, an attempt at self-destruction was actually made by the patient. Not less than seven patients attempted suicide by strangulation. One of these patients was found with a rope round her neck, and with a razor in one hand and a carving-knife in the other. This patient also made a desperate attempt to jump out of the window. Five patients tried to cut their throats ; one succeeded in her attempt without offering any explanation ; another endeavoured to account for the act by saying she thought she had killed her child, which had died from natural causes ; a third severed the larynx, and a fourth the trachea, under the impression that they had committed some great sin. This last patient had, for some weeks previous to committing the act, repeatedly told her friends that she would destroy herself. Another patient made a sort of half attempt by scratching her throat with a knife, and another stabbed

herself under the breast. Several took poison with the intention of destroying themselves. One swallowed some ammonia, having previously informed her relatives that she wished to be out of the world ; another tried to take laudanum, but was caught in the act and prevented from doing so. This patient had been discovered secreting poison about her person on seven different occasions. One patient attempted to leap from the window; another tried to set fire to her dress ; and a third, being determined to invent some new method, attempted to choke herself by thrusting her fingers down her throat.

It has been thought as well to dwell at some length on this point, as it is usually very easy to prevent any desperate attempt if the most ordinary precautions are observed by the relations.

Religious anxiety.—Many of the patients were troubled with doubts on religious matters. Several of them said that their souls were lost, and accused themselves of various sins, such as theft and murder. One patient affirmed that her evil deeds were the cause of all the deaths in the neighbourhood ; and another, that she had committed the unpardonable sin. A third stated that all were to be condemned, and prophesied that many sudden deaths were about to occur in the village ; a fourth accused herself of having been in constant communication with Satan ; a fifth declared she had been in hell for three nights. Several were in the habit of falling on their knees in the wards and asking the doctors and others to forgive them their sins ; another, less humble, stated that she had died to save mankind.

These symptoms are only exaggerations of the extreme religious views sometimes entertained by sane women at the change of life.

Extreme fear.—In a few cases there was an excessive dread of everything and everybody exhibited. Some of these patients cowered down in an attitude expressive of the greatest alarm, and started convulsively at the slightest noise. Others wandered about crying and wringing their hands, refusing to be comforted. In some this state of terror was coupled with delusion, as that the weight of the world

was left on their shoulders, that they were in flames, or that the house was on fire, or that everything was going wrong with them or their families.

Groundless suspicions.—Suspicion of friends and relations was frequently a prominent symptom. Sometimes these suspicions were of a very vague and undetermined character; but, in other cases, a distinct accusation was made. Many imagined that some unknown persons were plotting against them, and that they stood alone in the world, everyone else being opposed to them. One woman fancied that people were perpetually hovering around her with the intention of doing her some grievous injury; a second accused her friends of making up stories against her, and of wishing her out of the world; a third imagined that her master intended to murder her; a fourth, that she and all her family were about to be destroyed. Others believed that people continually followed them and insulted them, and these fancies occasionally led to strange scenes in the public thoroughfares.

In others the delusions assumed a more definite character. Many accused their relatives of attempting to poison them, and refused food in consequence. One woman thought poison had been administered to her in some pills, and that all the drinking-water in the neighbourhood had been tampered with. Another fancied that her enemies purposely kept her awake at night, and that some drug was thrown into her bedroom to make her sleep to wake no more; a third that persons were about to burn and stone her; a fourth that the house was undermined and about to fall on her; a fifth that spies surrounded the place at night; a sixth that she could not live or breathe if her friends were in the room; and a seventh, possibly not without reason, accused her husband of conspiring with all the hawkers and organ-grinders in the neighbourhood to torment her.

All these symptoms can be traced in the sane woman at the change of life, and are represented by the love of scandal and mischief-making, the readiness to find fault with others, and to take offence at imaginary insults so common in ladies at this critical epoch.

Violence to others.—The forms in which obnoxious symptoms showed themselves were in a few cases somewhat unusual. One woman perpetually spat in the faces of her friends; a second paced up and down the street with a knife in her hand, threatening the neighbours; a third took up a poker to her husband; a fourth wished to kill all her relatives; a fifth drove her children from the house with a knife; and a sixth threatened to crucify her nephew.

These peculiarities are represented in the healthy woman at the climacteric by the garrulousness, irritability of temper, and violence of language not uncommonly met with even in the higher classes of society at that interesting period.

Dementia.—When dementia resulted after all this mental perturbation, it sometimes took the form of that complete stolid silence so often seen in puerperal insanity, or else the language became very incoherent. It was worthy of remark that, even after the most profound dementia had existed, the patients frequently recovered.

Delusions.

Delusions of various kinds were exhibited as the disease advanced. These frequently assumed the form of delusions of suspicion as before noticed. Occasionally the patients imagined that some wrong had been actually committed against them. One woman fancied that she had been defrauded of all her money; another that she had had all her clothes stolen from her; and a third that some persons had torn her inside out. In another group of cases the delusions took the form of self-accusation, as that the patient had poisoned her children, that her character was gone, that she had been unfaithful to her husband, or that through her own fault she was ruined. One woman had several homicidal delusions. She thought her husband wished to destroy her, and also that she was ordered to murder somebody. To gratify this propensity she had killed a cat, and had insisted on burying it in her garden, near a chapel.

Another group of delusions referred to some supposed abnormal condition of the patient's body, as that she was

always on fire; that she was bewitched and would never recover her health; that her stomach was full of devils, who devoured all the food she swallowed; that spirits lived within her mouth, and controlled all her words; that her head was blown out with air, like a paper bag; that she had neither heart nor lungs; or that she had no personal existence, and was dead.

Delusions of pride were rare. There were only two cases of general paralysis found associated with insanity at the ménopause. One of these was too demented to have any delusions. The other had some quaint fancies, as, that she was a great dragon and as strong as Samson. Under this head came also the following delusions : that the patient had considerable property; that her brother was Lord Mayor of London ; that she possessed a magnificent voice; that she could see all over the world ; and that the Queen was going to keep her to prevent the devil getting at her.

In three married women, who had no children, delusions of a sexual character presented themselves. One declared she had had twenty children, another that her infant had been stolen, and a third that she was the Virgin Mary.

Hallucinations were not common. One woman affirmed that the wind was constantly talking to her; another that her dead mother appeared at her bedside ; a third had seen devils in the shape of dogs sitting on her bed, shaking their heads at her ; a fourth had seen spirits and stars constantly flying about around her; a fifth had been troubled with flashes of light passing before her eyes, and with nauseous tastes in her mouth; and a sixth had felt as if her brain was passing out through her ears, had jumped up from the pillow fancying she heard it escaping, and had also felt that her eyes were being drawn to the back of her head by some supernatural agency.

Prognosis.

The prognosis in insanity occurring at the change of life is decidedly favourable, 40 per cent. of the cases detailed above having been discharged, recovered. When it is considered that, as life advances, the probability of recovery in

most forms of insanity is notably diminished, the above percentage justifies our looking upon cerebral disorder connected with this period as fairly curable.

Course of the Disease.

Of the 40 cases that recovered, 3 only were discharged from the asylum in less than three months.

Of those who were kept in the asylum more than 3 months, 9 recovered in less than 6 months, 9 under 12 months, 8 under 24 months, 7 under 36 months, and only 4 recovered who were in the asylum more than 48 months.

From the above data, we may conclude that a quick recovery must not be looked for, and that restoration to health after a long period of mental aberration is also not to be anticipated.

The greatest number of recoveries took place in cases the duration of which was more than 3 months or less than 3 years.

The quickest recovery took place in 1 month, and the slowest in 4 years and 2 months, but these must be regarded as very exceptional cases.

The average duration of the disease in the 40 cases which recovered was 14 months and 12 days, or a little more than a year.

Twelve patients died in the asylum, and the remainder were still on the books when this enquiry was being made.

ANATOMICAL, PHYSIOLOGICAL, AND PATHOLOGICAL

INVESTIGATION OF EPILEPSIES.

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EPILEPTIC AND PARALYSED.

In the investigation of Epilepsies, or of any kind of case of nervous disease, we have three lines of investigation. We have—

1. To find the Organ damaged (Localisation).
2. To find the Functional affection of nerve tissue.
3. To find the alteration in Nutrition.

There is, in brief, (1) Anatomy, (2) Physiology, (3) and Pathology [1] in *each* case. The third part of the enquiry will be only incidentally considered. (See p. 326.) It is a very distinct part. As to the first, I shall speak of the localisation of lesions in epilepsies scarcely more than is necessary to illustrate the second, that is the physiological, part of the

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[1] This division will be clearer if I anticipate the definition to be given (p. 331) of an Epilepsy—an occasional, sudden, excessive and rapid discharge of grey matter of some *part* of the brain. The functional alteration (No. 2) is plain, for the very existence of repeated paroxysms tells us that there is a ʻ discharging lesion.' Under Anatomy (No. 1) we try, from a study of the paroxysm, to find where the discharging lesion is—to localise. And under Pathology (No. 3) we try to trace the steps of the abnormal process of nutrition by which the discharging lesion resulted. We have also a fourth heading for ʻ causes' (fright &c.) determining the discharge.

investigation. The reason is, that on the simplest 'epilepsies,'[1] which have to be first studied for principles of localisation, I have already written very many times.

There is one matter, however, which must be alluded to here.   In these simple cases of convulsion there is well exemplified an important principle, which, I presume, applies to all symptoms of the *cerebral* series.   This principle is of essential importance in unravelling the complexities of cases where mental symptoms occur after epileptic discharges.   It will also, I trust, help us to a more realistic view of that important symptom, loss of consciousness.   The principle is that those parts are wont to suffer first and most which serve in the more voluntary (special) operations, and those last and least which serve in the more automatic (general) operations.   Briefly to illustrate this, I quote from a paper I published in the 'Lancet' (February 1, 1873).   The quotation refers only to the mode of onset of the fits.   *The mode of onset is the most important matter in the anatomical investigation of any case of epilepsy.*

'There are three parts where fits of this group mostly begin : (1) in the hand; (2) in the face, or tongue, or both; (3) in the foot.   In other words, they usually begin in those parts of one side of the body which have the most voluntary uses.   The order of frequency in which parts suffer illustrates the same law.   I mean, that fits beginning in the hand are commonest; next in frequency are those which begin in the face or tongue, and rarest are those which begin in the foot.   The law is seen in details.   When the fit begins in the hand, the index-finger and thumb are usually the digits first seized ; when in the face, the side of the cheek is first in spasm ; when in the foot, almost invariably the great-toe.'

[1] I had written out for this paper some account of the paroxysms of these simplest cases (convulsions beginning unilaterally), but I withhold it, as its publication would be merely recapitulating what I have already said many times before.   I refer the reader to a paper, 'Study of Convulsions,' in the 'St. Andrew's Reports,' vol. iii. 1870 ; and for a summary to the report of a 'Case of Epileptiform Seizures, beginning in the Right Hand' ('Med. Times and Gazette,' December 23, 1871).   See also the *second* edition of the *second* volume of Reynolds' 'System of Medicine,' art. 'Convulsions' (*Class* 1.   *The Convulsion begins Unilaterally*, p. 277.)

The fits begin, it will be observed, in those parts which suffer most in the common form of hemiplegia.

It is very interesting to me to find that Ferrier's independent researches confirm the general principle above stated, so far as experiments on lower animals can be supposed to be comparable with the experiments disease makes on man.[1]

The following remarks will show the bearing of the above statements of the mode of onset of convulsive seizures : on the principle mentioned. In this group of fits the spasm ' prefers,' so to speak, to begin in those parts which have the more voluntary uses ; in other words, in those parts which have the more leading, independent, separate, and varied movements ; in other words still, in those parts the movements of which are last acquired (' educated '). In strict physiological definition, a voluntary part—the hand, for example—is one which has the greater number of *different* movements at the greater number of *different* intervals ; shortly, the more ' varied ' uses. An automatic part—the chest, for example—is one which has the greater number of similar movements at the greater number of equal intervals ; shortly, the more ' similar ' uses. Hence, convulsions which begin in the hand usually begin in the thumb and index-finger—in the most ' voluntary ' parts of the whole body.

It is not supposed that there are abrupt demarcations betwixt the two classes of movements. Movements of the shoulder (intermediate betwixt the arm and the thorax) may be considered as ' voluntary ' when compared with those of the chest, and automatic when compared with those of the

[1] The part I have italicised in the fourth of Ferrier's conclusions (which I give below from the ' Brit. Med. Journ.' April 26, 1873) is what I especially refer to.

(4). The proximate causes of the different epilepsies are, as Dr. Hughlings Jackson supposes, 'discharging lesions' of the different centres in the cerebral hemispheres. The affection may be limited artificially to one muscle, or group of muscles, or may be made to involve all the muscles represented in the cerebral hemispheres, with foaming at the mouth, biting of the tongue, and loss of consciousness. When induced artificially in animals, the affection, as a rule, *first invades the muscles most in voluntary use*, in striking harmony with the clinical observations of Dr. Hughlings Jackson.

(5). Chorea is of the same nature as epilepsy, dependent on momentary discharging lesions of the individual cerebral centres. In this respect, Dr. Hughlings Jackson's views are again experimentally confirmed.

arm. To illustrate : it is just as a metal is positive or negative, according as it is considered in relation to the one before it or to the one after it, in the electro-chemical series.

Each of the three varieties of fits is supposed to depend on discharge of more or less of the sensori-motor processes of some particular series. The mode of onset gives us a clue to the series. Thus, those fits which begin in the hand are supposed to depend on discharge of a part of the cerebrum where the hand is largely represented in movements to which other movements (those of the face and leg) are sequent and subordinate. Again, in fits beginning in the foot, the internal part discharged contains processes for certain movements of the foot and lower limb, to which certain other movements (those of the upper limb) are sequent and subordinate. In *this* series the foot is the voluntary (special, &c.) part. Hence the remark that the mode of onset is the most important matter in the paroxysm for our consideration. It points to the part of the brain which discharges, or where the discharge begins.

As a more definite illustration, I will mention that in fits beginning in the (right) cheek, there is often temporary defect of speech. The fit (for it usually, the patient tells us, affects the tongue also) begins in the parts for the exteriorisation of speech. There is, *after* some of these seizures, epileptic aphasia,[1] analogous to epileptic hemiplegia.

The main object of this article, however, is the topic to which I now come, viz. the Physiology of Epilepsies—the second line of investigation.

As I would define it in its application to the investigation of Epilepsies, Physiology is concerned with the *function* of nerve tissue. What I call a ' discharging lesion ' is *one* of the two morbid modifications of the function of this tissue. It will, however, be impossible to limit the remarks rigidly to this line of investigation. It will be necessary to consider

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[1] After some fits, beginning in the hand (right), there is occasionally temporary defect of speech.

not only the discharging lesions themselves, but, for illustration, we must speak of the different phenomena (different kinds of seizures) which differently *placed* 'discharging lesions' produce—of the localisation of the discharges, a topic which strictly belongs to the Anatomical division of our enquiry. Our particular task, however, is the consideration of a certain functional change in nerve tissue.

Before I speak of abnormal functional *changes*, I must expressly point out that I do not use the word 'functional' in senses in which it is frequently used. It is sometimes used as a name for minute changes, or for those the existence of which we are obliged to infer because nervous symptoms are present, but which we do not expect to discover *postmortem*. For instance, it is said that epilepsy and chorea are functional diseases, it being meant that the changes on which the symptoms depend are so slight that they do not involve alteration of structure, but only of function. This is, I think, an inconvenient way of using the word. The real meaning is little more than that the changes spoken of are as yet undiscovered. In the second class of what I shall call functional changes the changes are slight, but their slightness is no essential part of the definition to be given.

The term is used more loosely still. Thus, when a patient has a transient and imperfect paralysis—for instance, slight hemiplegia—lasting a day or two only, the internal changes may be declared to be functional *simply because the external symptom presented was a slight and transitory one.* In reality, the slightness and transientness of a paralytic symptom depend on the *extent* of the lesion of the nervous organ, not on the *nature* of the affection of nerve tissue of that organ. If there be a limited lesion the patient will recover quickly ; and his recovery does not always follow, if indeed it ever does, because the damage to the motor tract is repaired, but simply because he has lost only a small *quantity* of that tract. For it is manifest to those who make *post-mortem* examinations that recovery from paralysis will occur when a part of the motor tract is permanently wanting. Nor will this be strange to those clinical observers who *do not* make *post-mortem* examinations, if they consider the facts bearing

on the plan of structure of nervous organs (Appendix II.), and that when recovery follows in a case of paralysis it observes a particular order—it is from that of the more automatic parts to that of the most voluntary. Recoverability from paralysis is chiefly a question of the *size* of the lesion— quantity of nerve tissue destroyed. As lesions differ in size in all degrees—for example, lesions produced by clots from the size of a pea to that of a hen's egg—there are all degrees of paralysis and all degrees of recoverability. There is no need to explain a transient paralysis by a state of nerve tissue. Transientness of paralysis cannot be taken as evidence that nervous *structure* has not been *destroyed*. That parts of the cerebral hemisphere may be destroyed when there are no obvious or striking symptoms is well known. But it is so of the motor tract also. I shall have again to insist on this class of facts (Appendix I.).

Another objection to a not uncommon use of the word is, that under it two very distinct things are confounded, viz. functional changes and the pathological processes which lead to them. This is, I think, particularly unfortunate ; physiology and pathology are mixed. I will here remark on the difference of the two things by quoting from an article on 'Hemichorea,' I published in the ' Edin. Med. Journ.' Oct. 1868.[1]

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[1] One reason for quoting *that* article is, that I may correct a misapprehension under which most of those labour who have criticised my opinions on the pathology of chorea. The remarks quoted in the text, taken with the statement of the second division of my hypothesis on an earlier page of the article quoted from—' That this local instability is *frequently* brought about,' &c.—show that I have not undertaken to defend the doctrine that embolism is the *sole* cause of chorea (cause of the instability of nerve tissue in chorea). The fault, however, is my own ; I freely admit this. By a clerical error which I ought to have corrected, I am made, in the ' Mirror ' of the ' Lancet,' November 26, 1864, p. 606, to say ' limited *to* softening ' of the brain, instead of ' limited softening ' of the brain. I have, however, corrected it by implication. In a report of the Obstetrical Society (' Med. Times and Gazette,' August 1, 1868), I say : ' This instability, although *frequently* the result of anæmia [hyperæmia I now think] from blocking, *might doubtless be induced in other ways*, as in the choreiform movements which sometimes occur during recovery from epileptic hemiplegia, the secondary results of coarse disease of a cerebral hemisphere.' In the ' Lond. Hosp. Reports,' 1864, vol. i. p. 459, I write : ' I think, from many circumstances, that embolism is a *frequent* cause of chorea.' I believe, however, that embolism (or blocking of arteries by some process) is *almost* the sole cause of chorea. Nay,

' Just as loss of function—for instance, palsy—follows destruction of nerve tissue, *however produced*—by clot, by tumour, by injury, &c.—so *disorder* of function—(discharge) for instance, chorea or spasm—results from instability of nerve tissue, *however produced*—by mechanically produced anæmia (?), and, as I think, by embolism.' [1]   There is a still further objection to the common use of the word.

. I have for some years used the term 'functional' to describe the morbid alterations of *the normal function of nerve tissue.* Therefore, before I speak of these alterations, we must notice what the normal function of nerve tissue is.   Its function is to store up and to expend force.   It is true that this is the function of *all* organic matter, but it is *par excellence* the function of nerve tissue.   There are *but two* kinds of alteration of function from disease.   Saying nothing of degrees of each, there is on the one hand loss of function, and on the other over-function (not better function).   In the former, nerve tissue ceases to store up, and therefore to expend force.   In the latter, more nerve force is stored up than in health, and more is therefore expended ; the nerve tissue is highly unstable.   Under the first head come palsies (acineses), anæsthesiæ; under the second, chorea, epilepsies, tetanus (hypercineses), neuralgia, &c.   In a not uncommon way of using the word 'functional' no distinction is made; this is the further objection just mentioned.   The changes which produce paralysis (provided the symptoms be slight in degree or transitory), as well as the changes which produce tetanus, chorea, &c., are spoken of as being functional.   The term is, in short, used for two opposite states of nerve tissue.

I used to consider these two states of nerve tissue to be degrees of but one condition.   I will stay to remark on this point, although its consideration belongs strictly to pathology. The remarks will, however, scarcely be digressive, as two important facts will be incidentally disclosed which are re-

speaking in the very strictest sense, I do not believe that two *different* pathological processes would damage an organ in absolutely the same way; from two pathological processes leading to instability of convolutions near to the corpus striatum, I should expect slight, even if insignificant, differences in the movements resulting.

[1] The functional change of nerve tissue in epilepsies, in chorea, and in tetanus also, is supposed to be the same.

quired for a later development of the physiological part of our subject.

One reason I had for thinking these two states to be degrees of one condition was suggested by the very striking clinical fact that paralysis is frequently associated with convulsion. For example, hemiplegia not unfrequently follows convulsions beginning on one side; it is the epileptic hemiplegia of Dr. Todd. I now explain this remarkable sequence by the supposition that the paralysis is caused by the strong discharge in the fit. But here it must be mentioned that there is occasionally hemiplegia (partial hemiplegia) *before* the first fit, and when the attack is on we see that those parts which have been *paralysed* are first and most *convulsed*. This puzzles students. They think it contradictory that *paralysed* parts should be the very ones which are 'picked out' for spasm. And in trying to 'remember' on which side a patient was convulsed, a student will occasionally really *infer* the side convulsed, as is shown by his saying, 'It *must* have been his left side, *because his right side was paralysed.*' I used, as above mentioned, to explain the association by the notion that the condition of nerve tissue which caused the paralysis was a more extreme degree of the same condition as that which caused the spasm. But I think a more satisfactory explanation can be given (see Appendix II.).

To return now to the two kinds of functional changes. In the first class of cases nerve tissue is frequently actually destroyed, as when it is broken up by a clot; but, at any rate, its function is lost. In the second class nerve tissue is unstable, it energises too much—it discharges on slight provocation, and discharges strongly. It matters nothing for the definition how the destruction or instability be effected—by what pathological process. Destruction (loss of function) of nerve tissue may be the result of tearing up by blood clot, of slow wasting (as in spinal amaurosis), or it may probably be the result of a strong discharge, as in cases of epileptic hemiplegia (*vide supra*).[1] Then as to instability; it may, I believe be produced by hyperæmia consequent on blocking of vessels

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[1] As here implied, one does not mean necessarily physical destruction, nor *permanent* loss of function.

(see p. 328), by 'irritation' of a tumour, and probably in many other ways.

The mode of *production* of the discharging lesion (of unstable nerve tissue—grey matter) belongs to pathology. Something will be said under this head (pp. 326–8).

Using somewhat curiously compounded but convenient terms, the two kinds of functional changes may occasionally be called ' destroying lesions ' and ' discharging lesions.'

Epilepsies are the results of the second class of functional changes ; they are, speaking briefly, 'discharging lesions.' But there are many varieties of discharges. Defined from the paroxysm, an epilepsy is a *sudden, excessive,* and *rapid* discharge of grey matter of some *part* of the brain ; it is a *local* discharge. To define it from the functional alteration, we say there is in a case of epilepsy grey matter which is so abnormally nourished that it *occasionally* reaches very high tension and very unstable equilibrium, and therefore occasionally 'explodes.' The two definitions are of different faces of the same thing.

It will be observed that the ' discharging lesion ' of epilepsy is supposed to be a *permanent* lesion ; there is grey matter which, since it is permanently under conditions of abnormal nutrition, is permanently abnormal in function. That this permanent abnormality is a varying state has been said ; it has been remarked that the grey matter *occasionally* reaches high tension, and therefore *occasionally* discharges (or is discharged). There are waves of stability and instability. It follows from this that the first fit is supposed to be a discharge of a part which has *for some time* before been in a state of mal-nutrition ; and a still further inference is, that such ' causes ' of epilepsies as fright are only determining causes of the *first* explosion. Many of the ' premonitory symptoms ' of a first attack are probably results of slight discharges ; [1] they are miniature fits.

There is not, I think, nowadays much difficulty in un-

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[1] The other day I congratulated a mother on the fact that her son had not had a severe fit. She, however, regretted it, saying that the severe fit 'cleared his system,' whilst the slight fits rendered him, from their frequency, unable to go to business.

derstanding that from a *permanent* abnormality of nutrition there can result *occasional* disorderly exhibition of function. The process in epilepsies roughly corresponds to what is supposed to occur normally in the ganglia of the heart; there is, it seems to me, only a particular application of certain physiological doctrines long since put forward. Thus, I quote from Baker's edition (seventh) of Dr. Kirkes' 'Physiology' (p. 141): 'Why these nervous centres' [nervous ganglia of the heart] 'should issue impulses for rhythmic rather than for continuous action, is still a debateable point. The most philosophical interpretation yet given of it, and of rhythmic processes in general, is that by Mr. Paget, who regards them as dependent on rhythmic nutrition—i.e. on a method of nutrition in which the acting parts are *gradually raised*, with time-regulated progress, to a certain state of *instability of composition*, which then issues in the discharge of their functions, *e.g.* of nerve force in the case of the cardiac ganglia, by which force the muscular walls are excited to contraction. . . . *All organic processes* seem to be regulated with exact observance of time ; and *rhythmic nutrition and action*, as exhibited in the action of the heart, are but well-marked examples of such chronometric arrangement.' (No italics in original.) Schrœder van der Kolk writes, as his fourth conclusion on epilepsy, that 'The special seat and starting-point of these convulsive movements is situated in the ganglionic cells of the medulla oblongata, which, as reflex ganglia, possess the peculiar property that, when once brought into an excited condition, they may more or less suddenly discharge themselves and communicate their influence to different nervous filaments. After their discharge a certain time is again required to bring them to their former degree of excitability, and to render them capable of fresh discharges, just as we see to be the case with electric batteries, or in the phenomena of an electrical fish.'[1]

So long as a patient has one-sided *palsy*, we do not doubt him to have a 'destroying lesion' of his 'motor tract.' Similarly, so long as a patient has recurrences of one-sided

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[1] 'Syd. Soc. Trans.' p. 283.

convulsion, so long has he, I believe, a ' discharging lesion ' of convolutions near to the motor tract.

Of course it is not denied that *healthy* nerve tissue can be made by certain very abnormal provocations to discharge so strongly as to produce convulsions. Thus, convulsions occur during rapid tearing up of nervous substance in large hæmorrhages, either when the bleeding is in the cerebral hemisphere or in the pons Varolii. They are producible in healthy animals by *rapid* bleeding to death, as Kusmaul and Tenner's experiments show. Besides, there are the very striking results of direct action on the brain in the experiments of Fritsch, Hitzig, and Ferrier. I speak of cases in which fits are either one-sided, begin by a very local aura, or of cases where the fits recur.[1] In these cases there must be a local and persistent lesion.

Symptoms of instability in disease are, I suppose, an exaggeration with caricature of the effects of healthy discharges. Of some this is easily seen. In chorea the movements are often only awkwardnesses—only slight departures from health (see p. 183); the poor child is occasionally beaten by his parents for clumsiness. The following remarks, referring to healthy discharges, would, with a few slight modifications, apply to some of the trifling degrees of instability of disease. ' The longer repair goes on unopposed by appreciable waste, the greater must become the instability of the nerve centres, and the greater their readiness to act; so that there must at length come a time when the *slightest impressions* will produce motions.'[2] In fact, there is at the time Spencer is speaking of, a healthy and yet random discharge. ' On awakening from refreshing sleep,

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[1] If we are called to a patient in his first convulsion, and if that convulsion be ' general,' we often cannot tell to what it is owing; whether, for instance, it bo owing to some local cause, acting on a previously healthy brain—as rupture of an intracranial aneurism—or be owing to explosion of some part of the brain which has long been getting into a highly unstable condition. I do not deny that it is easy to guess; we should mostly guess rightly. All I mean, is that we do not make a diagnosis worth calling one if, when we are called to a person who has had a severe convulsion, and of whose case we have no history, we turn out to bo right in having said, ' It is a case of epilepsy.'

[2] Spencer, ' Psychology,' vol. i. p. 90.

there commonly occurs an involuntary stretching of the muscles of the whole body, showing an immense undirected motor discharge.'[1]   Then a sneeze is a sort of healthy epilepsy.

Before I close the remarks on the functional nature of the lesion in epilepsies, I will consider briefly the pathology of discharging lesions in general, taking as a starting-point the remark made on page 321, that I used to believe the two functional states of nerve tissue were degrees of one condition.   At that time, following Radcliffe, Handfield Jones, and Anstie, I considered that the nutrition of nerve tissue was *imperfect* in unstable nerve tissue.   In a certain sense I think so still.   Recently, indeed, I have been led to think that the view I now hold is in some respects simply a modification of the one they have long taught.   I say this however, to acknowledge an obligation.   The reader will, of course, judge of the views of these physicians from what they have written themselves, and hold me responsible for what I now write.

There are, I submit, two ways in which nutrition may be imperfect—in quantity and in quality.   I believe that nerve tissue in discharging lesions is over-nourished in the former sense and worse-nourished in the latter.   In order to make my meaning clearer, I will take chemical illustrations and use chemical nomenclature.   Two bodies may be of the same Constitution, but yet of very different Composition. For example, the constitution of acetic acid and of the chloracetic acids is the same ; but they differ in composition, as in the latter hydrogen has been replaced by chlorine. The ' structure,' however, is unaltered.[2]

I believe, then, that the highly unstable nervous matter of disease (in a ' discharging lesion ') differs in composition, but not in constitution, from the comparatively stable grey matter of health.   The alteration in composition is of course such that the nervous substance formed is more explosive.

[1] Spencer, *Op. cit.* vol. i. p. 90.

[2] Perhaps a better illustration may be the formation of that highly unstable substance, gun-cotton, from stable cotton by the process of substituting three atoms of hydrogen by $N O^2$.

We must suppose that there is some order in this Substitution-Nutrition, and we must infer that it is in the direction of explosiveness, or instability. The following is a speculation as to the kind of alteration of composition. One striking constituent of nervous matter is phosphorus. It belongs to the chemical class of triads of which other members are nitrogen and arsenic. My speculation is that in the abnormal nutritive process producing unstable nervous matter the phosphorus ingredient is replaced by its chemical congener, nitrogen. There is a substitution compound; the replacement probably occurs in different degrees, as it does in the three differing chloracetic acids. If nitrogen be substituted, as supposed, we can easily understand that the substance produced would be more explosive. The supposed value of arsenic in certain nervous affections is significant; it is another member of the group of triads. The nutrition is, therefore, assumed to be defective not in quantity but in quality, in those functional alterations I call 'discharging lesions.'

I think these speculations have a practical bearing on the dietetic treatment of epileptics. I advise epileptics not to eat much meat (not much nitrogenised food), nor, indeed, much of anything. I was, however, led to give this advice in another way, not by the speculation just mentioned, but by the following remarks of Dr. Haughton:—

'The hunted deer will outrun the leopard in a fair and open chase, because the force supplied to its muscles by the vegetable food is capable of being given out *continuously for a long period of time*; but in a sudden rush at a near distance the leopard will infallibly overtake the deer, because its flesh food stores up in the blood a reserve of force capable of being given out *instantaneously* in the form of exceedingly *rapid* muscular actions.'[1]

I think a great part of the 'weakness' and languor of some of those persons who suffer what is popularly called 'nervous debility,' and who are often hypochondriacal, is explainable on the supposition that their nervous tissue is

[1] 'Address on the Relation of Food to Work,' p. 28. No italics in original.

over-nourished in quantity, and yet so imperfectly nourished
in quality that it is explosive; or, let us use in this simple
and not uncommon condition of ill-health, the expression,
more irritable nervous matter. They often keep up this
irritability by frequent eating and drinking. My colleague,
Dr. Andrew Clark, insists that the most successful treatment
of such persons is putting them on a very simple unstimu-
lating diet, alcohol in particular being forbidden. My spe-
culation is, that the good results are owing to the formation
of less explosive or less irritable nervous substance—one of a
more normally stable composition. The excitability of these
patients reminds one of the fact that the nerves of a weak
animal are found, experimentally, to conduct with greater
velocity than those of a healthy one.

The above speculations seem to me to harmonise with
certain doctrines on the *pathology* of discharging lesions in
some cases. Thus, I believe that chorea frequently, and
epilepsies sometimes, are produced by embolism or throm-
bosis. The cases spoken of (p. 322), of convulsions *fol-
lowing* hemiplegia, and affecting first and most the parts
already partially paralysed, are, I believe, cases in which
there is usually plugging of branches of the middle cerebral
artery. That plugging of vessels can lead to local *destruction*
of nerve tissue is plain enough. But I think there is a
strong presumption that it can lead to *instability* of grey
matter. When (as stated p. 321) I held the doctrine that
the two functional states of nerve tissue were degrees of but
one condition, I was under the mistaken impression that
plugging of vessels led always to local anæmia. But it can
lead to local hyperæmia : this bears most significantly on
the production of unstable nervous matter. It is to be ob-
served that, though there is in hyperæmia thus caused *more*
blood, there is less change of blood ; hence the nutrition will
be of a more general kind. That there is less change of
blood is a very important departure from the normal circu-
lation of the brain, as one peculiarity of this organ is that
a large quantity of blood passes *through it* ; it has a great
change of blood.

I may most conveniently state the further points on this

matter by the following quotation from a paper I contributed to the 'Medical Times and Gazette,' March 6, 1869. Although it was written *à propos* of chorea, it applies to the discharging lesions of other symptoms (epilepsies &c.) :—

'It has been often urged as an argument against embolism being the cause of chorea, that *anæmia* from plugging of vessels can scarcely lead to *increased* expenditure of force. If arteries be plugged, it seems certain that the nutrition of parts they supply will be *defective* ; still, it does not follow that it will be *decreased*. For, according to certain physiological experiments, it seems that plugging of a small artery does *not* always cause *anæmia* of the capillary region to which the vessel should deliver arterial blood. On the contrary, it may cause *congestion,* and may even lead to extravasation. I must, for the facts and arguments of this question, refer the reader to MM. Prévost and Cotard's work, " Études physiologiques et pathologiques sur le Ramollissement cérébral " (Paris, 1866), and especially to a section (p. 38), " De la congestion qui accompagne infarctus." I will only quote the last of the three conclusions from their experiments (italicising some words) :—" Consécutivement aux *oblitérations artériales* il se produit habituellement de *l'hyperémie* et de la tuméfaction," &c. Dr. Ivan Poumeau has also published very interesting statements on the effects of plugging of vessels—" Du Rôle de l'Inflammation dans le Ramollissement cérébral," 1866. To explain how increase of blood results from *blocking* of an artery is a very difficult thing. The first step towards an explanation lies in determining whether the increase of blood is of the venous or arterial kind. Rokitansky thinks the local congestion is produced by increased pressure on the collateral arteries ; Virchow thinks it owing to return of blood from the veins. It is impossible to decide without further evidence when such men differ, except, perhaps, by the clumsy expedient of fitting the two views together—viz. that in the periphery of the congested spot there is arterial congestion, and in the centre venous congestion. As it seems to me, the local increase in quantity of blood, at all events, has an important bearing on the production of chorea *and other symptoms*

implying increased expenditure of force. If it be venous, we may suppose that, although nutrition may be carried on faster, it will lead to *more imperfect and more easily decomposable nervous matter*; or, if we suppose the nerve force is supplied from the blood to the nervous structure in the same way as recent investigators believe force is supplied to the muscles, the increase in the quantity of blood is still significant when associated with increased expenditure of force. I suppose we may fairly say that the general character of blood, which is stagnant or slowly changed, will be venous rather than arterial.'

I believe then, as Dr. Russell Reynolds says, that ' The proximate cause of convulsions is an abnormal increase in the nutritive changes of the nervous centres ;' but I also think that the nutrition is carried on on an inferior level. I suppose that if the nervous matter cannot get enough phosphorus, it takes nitrogen, provided it is under conditions favourable to some kind of nutrition—just as a plant will take soda when it cannot get potash.

Let us now consider ' discharging lesions,' with a view to a more formal definition of epilepsy in the sense in which I use the term in this paper. It will, however, be convenient to state first what things are not essential to the novel definition.

First. Epilepsy is not one particular grouping of symptoms occurring occasionally ; it is a name for any sort of nervous symptom or group of symptoms occurring occasionally from local discharge. Whether the discharge puts muscles in movement or not—that is, whether there be a *convulsion* or not—matters nothing for the definition. A paroxysm of ' subjective ' sensation of smell is an epilepsy as much as is a paroxysm of convulsion ; each is the result of sudden local discharge of grey matter.

Secondly. It does not matter for the definition whether there be Loss of Consciousness or not ; loss of consciousness is a fundamental thing in most of the accepted definitions. If there be no loss of consciousness, there is, according to most physicians, not epilepsy, and then the term ' epileptiform ' is used. But even when using the term epilepsy in the or-

dinary sense of the word the separation into cases where there is and where there is not loss of consciousness, has no *physiological* warrant. It is an arbitrary distinction of psychological parentage. Loss of consciousness is not an utterly different thing from other symptoms. It is not to be spoken of as an epiphenomenon, nor as a complication. Consciousness has, of course, anatomical substrata as much as speaking has. The sensori-motor processes concerned in consciousness are only in degree different from others. They are the *most* special of all special nervous processes, the series evolved out of all other (lower) series.

To lose consciousness is to lose *the use of the most special of all nervous processes* whatsoever.[1] If those parts of the brain be first affected by strong discharge where the most special of all nervous processes lie, there will be loss of consciousness *at the outset.* If processes of a subordinate series be discharged, loss of consciousness of course occurs later. For example, in cases of convulsions beginning in the hand, consciousness is in most cases lost as soon as or just before the leg is reached by the spasm. In these cases the internal process will be that consciousness is lost as soon as the most special of all processes *are reached* by the internal discharge, or (since the sensori-motor processes underlying consciousness are evolved *out of* lower series. See Appendix II.) when a large quantity of a subordinate and yet important series is put *hors de combat.*[2] But, of course, one does not locate consciousness so geographically as the mere words we must use seem to imply. If a patient suddenly loses by any process the use of *any large part* of either of the two *highest* divisions of the nervous system, he will lose consciousness.

Epilepsy is the name for occasional, sudden, excessive, rapid, and local discharges of grey matter.

That the epileptic attacks are *sudden,* needs no showing;

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[1] I say to ' lose the use,' because the use of sensori-motor processes will be temporarily lost *during discharge,* causing their excessive excitation as much as it is permanently lost when they are implicated by gross lesion.

[2] Much depends on the rapidity of the discharge ; the more rapid the spasm of the arm the sooner is consciousness lost. It is not to be inferred from the foregoing that other factors, as asphyxia, are not concerned.

that the discharge is excessive is plain enough in those cases where convulsion occurs; and in all cases the inference is irresistible that the morbid discharge is far greater than the corresponding healthy discharge. In those fits which begin in the hand the discharge is so strong that the pain from the cramped muscles is very great.

It is convenient to speak next of the *occasional*, or paroxysmal occurrence of epileptic discharge. There are discharges which are *not* occasional; and thus the word ' occasional,' in my definition of epilepsy, excludes the ' interrupted continuous' discharges of chorea[1] and the almost uniform stream of discharges in tetanus.

The next point is as to the discharge being *local*. I will quote[2] Voisin, to show that (using just now the term Epilepsy in its commonly accepted sense) epileptic attacks in the same patient are stated by one good authority to be like one another. If so, the discharge in each fit must be in the very same internal part; yet attacks differ in different patients, and thus the internal discharges causing them must differ in locality.

At page 595, *Op. cit.*, Voisin writes :—

' Axenfeld a fait remarquer que les attaques se reproduisent le plus souvent avec une uniformité absolue, avec leurs auras, leurs caractères propres et leurs complications. Il s'établit une sorte d'habitude d'après laquelle tel individu

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[1] I would strongly urge that it is as important to note differences in the *time rates* of discharges, as in the specialty of the movements those discharges develope. For example, the dog's chorea differs from that of the child, not only in that the former is almost a repetition of *one* movement, whilst in the latter there are mostly successions of *different* movements, but also in that the choreal movements in the dog are nearly rhythmical, whilst in the child the intervals of the movements are varied as the movements are.

[2] ' Nouveau Dictionnaire de Médecine et de Chirurgie Pratiques, Art. Épilepsie.' This is an article of great value.

I give Voisin's definition of epilepsy :—' L'épilepsie est bien une maladie et non pas un symptôme, malgré ce que peuvent dire certains auteurs, qui ne me paraissent pas avoir suffisamment vécu au milieu d'une population d'épileptiques ' (Voisin, *Op. cit.* p. 581). He thus defines epilepsy : ' Profitant des travaux de ces maîtres (Trousseau, Th. Herpin, B. A. Morel), nous pensons donc que l'on doit définir l'épilepsie une *maladie chronique, apyrétique, caractérisée par des attaques convulsives, des vertiges, des absences, qui frappent l'individu d'une façon irrégulière, au milieu de la santé, souvent, en apparence, la plus parfaite* ' (Voisin, *Op. cit.* p. 581).

sentira toujours la même aura, poussera toujours un cri et le même cri, tombera sur le même point du corps, le front l'occiput se blessera de la même façon, présentera les mêmes mouvements convulsifs, se mordra la langue au même point, se luxera toujours une même épaule, sera toujours pris de délire après l'attaque.'

This being so, those who hold that the medulla oblongata is the part which discharges in epilepsy, have, as well as others, to note *paroxysms* with great care, in order to find out how it is that in one patient there are fits of a certain kind, and in another of another kind, however much the seizures in the two patients may agree in the most fundamental characteristics required by the ordinary definition of epilepsy.

But, as stated, numerous and very different nervous symptoms may be epileptic in my definition of the term. And as any part of the grey matter of the cerebrum may become unstable, there will be all varieties of epilepsy, according to the exact position—according to the extent of the grey matter altered—and there will be all degrees according to the degree of instability. But, for illustration, we shall give a list (see next page). I wish first to speak of cases of limited seizures, or, if the expression be preferred, of partial seizures. I wish to point out that we must not take *degrees* of epilepsies for *varieties* of epilepsies.

It is obvious that there are all degrees of epilepsies. A fit which is limited to the hand is not a fit of a different *kind* from one which begins in the hand, and then *spreads* all over the body, and is, after a certain stage, 'attended by' loss of consciousness. The latter seizure is but the result of a much stronger discharge. A fit which begins in a part like the hand, and *becomes* universal, *is* different from one in which the convulsion is nearly universal at the very first; a different area of grey matter must be discharged in each. Another illustration as to degrees of seizures is that a patient may one day have transient loss of consciousness only, who, on another day, has this instantly 'followed by' convulsion. There are here again differences of degree only depending on the strength of the initial discharge. For, as

already remarked, the processes put *hors de combat* during the discharge which causes loss of consciousness, are not *fundamentally* different from sensori-*motor* processes, discharge of which produces convulsion. The former are highly special sensori-*motor* processes, evolved out of—as it were, continuous with—lower sensori-*motor* processes.

There will, however, be a compound result from increasing strength of discharge. We see, externally, that the stronger the spasm is the *wider* spread it is—the stronger the internal discharge the further it will spread. There are *two* ways of spreading. The discharge will not only explode healthy *lower* centres, but will probably spread, as it were, laterally, to healthy associated centres in the brain. My speculation is, that the lateral spreading is by arteries[1] and their vasomotor nerves ; the spreading is in Arterial Regions.

The following are Epilepsies :—

(1) A sudden and temporary stench in the nose, with transient unconsciousness. (2) A sudden and temporary development of blue vision. (3) Spasm of the right side of the face and stoppage of speech. (4) Tingling of the index finger and thumb, followed by spasm of the hand and forearm. (5) A convulsion almost instantly universal with immediate loss of consciousness. (6) Certain vertiginous attacks.

All these six seizures are alike in that each results from an occasional and excessive discharge of unstable grey matter. This is the one functional alteration of nerve tissue underlying the different phenomena.

The investigation does not end here; rather it begins here. The very general nature of the physiological statement *necessitates* most careful method in the anatomical part

[1] On Arterial Regions in the brain I have often written, with regard to the interpretation of epilepsies ; *one* factor in the internal change causing the paroxysm, being, I think, arterial contraction. See ' Lond. Hosp. Reports,' vol. i. 1864, p. 466. The scheme of arterial supply is, no doubt, as definite and as purposive as are the arrangements of nerve fibres and cells. Very little consideration shows, that the more special nerve centres become the more independent must be their nutrition, and this necessitates an increasing limitation of arterial areas. I believe the arteries have to do with developing *sequences* of movements. It is a matter of significance that the arteries of the cortical grey matter have no anastomosis; they are ' terminal arteries.'

of the investigation. For, obviously, all the six epilepsies, notwithstanding their *physiological* likeness, are very unlike *anatomically*—that is, in their Localisation. The area of grey matter altered in each must be in a different place, or the phenomena could not differ. In a word, although the functional alteration is the same in all epilepsies, the seats of those functional alterations are various.

No one can admit more fully than I do the difficulties in the Localisation of ' discharging lesions.' The very method I work on keeps the difficulties well before me. I know well that in most of the six cases I cannot localise the discharging lesion. But this failure is no objection to the method. I still urge that we should go on trying to localise; and we should, so far as is practicable, work in the same realistic manner as we do in cases of paralysis. Surely it is as important to localise ' discharging lesions ' as it is to localise ' destroying lesions.' Every physician attempts the latter. If a man be hemiplegic, we observe the region affected by palsy to find where the central destroying lesion is. But, in cases of convulsion, the region affected by spasm is scarcely ever methodically studied in order to find where lies the central destroying lesion. Some most practical-minded men show great interest in discussions as to the particular muscles of the face most implicated in cases of hemi*plegia*, but no zeal in investigating the particular muscles of the face first and most implicated in cases of hemi-*spasm* (convulsion beginning unilaterally). The question often put is, Is it a case of epilepsy? or is it only an epileptiform seizure? The question should be, Where is the discharging lesion in this case? The plan of studying cases of disease, so far as they show *approaches to certain clinical entities,* is, I feel convinced, an unfruitful one. To show that I have long held this view, I will quote from a paper I quoted from in my former article. The case referred to in the subjoined extract was one of amaurosis associated with convulsion beginning unilaterally :—

' And I ought here to remark more particularly that I do not say simply that amaurosis in Case XX. occurs with epilepsy. Not that this, according to a common use of

terms, would be a wrong phrase, but because the term "epi-lepsy" has not a sufficiently precise meaning. Besides, this expression has not vitality in itself. It is extremely impor-tant *to describe* the sort of fit an amaurotic patient may have; to call it "Epilepsy" gives the symptom too elastic a meaning, and careless people might think they had disposed of this part of a case by saying "the amaurosis is complicated with epilepsy." This classification would, I think, be an approach, under the cover of a conventional and yet an unreal pre-ciseness, to the analogous looseness of saying "the amaurosis is complicated with paralysis," leaving it doubtful what sort of palsy there was. We want positive information as to how a convulsion is a *departure* from health of muscles and mus-cular groups and health of nervous organs and tissues, and not as to how far it *approaches* our idea of the almost meta-physical conception "genuine" Epilepsy. . . . . there are to be found on record scarcely any positive statements of what has really happened in particular convulsive paroxysms —a process which sometimes occurs under our very eyes. I am fully aware that there are admirable accounts of the worst fits as types, but some of these accounts are descrip-tions more of dramas of great human interest, than calm and cold scientific observations in an orderly sequence of the outward phenomena of an inwardly suffering nervous system.'[1]

One fruitful method of study is by seeking symptoms re-sulting from 'destroying lesions'[2] which correspond to those the results of 'discharging lesions.' I will illustrate this method, but only in outline.

If we consider the effects of a destroying lesion of the corpus striatum, we find paralysis of the face, arm, and leg. There are paroxysms of convulsion which correspond, and which depend on discharges of convolutions near to this body;[3] we will illustrate by lesions of the *left* side of the brain.

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[1] 'Roy. Lond. Ophth. Hosp. Reports,' 1866.

[2] See 'Remarks on Hemi-kineses,' p. 178.

I say nothing of the tongue, as I have not been able (except in one case) to make any observations on it in convulsions.

First Degree.

Corpus Striatum Palsy.	*Corpus Striatum Epilepsy.*
Mouth turns to left.	Mouth drawn to right.
Right arm paralysed.	Right arm convulsed.
Right leg paralysed.	Right leg convulsed.

This is, however, only a comparison with the first degree of paralysis. A graver lesion of the corpus striatum produces the symptoms named in the next list, and there is a further degree of convulsion which corresponds.

Second Degree.

Corpus Striatum Palsy.	*Corpus Striatum Epilepsy.*
Head turns to left.	Head drawn to right.
Two eyes turn to left.	Two eyes drawn to right.
Face turns to left.	Face drawn to right.
Trunk muscles weaker on right.	Trunk muscles in spasm on right (?).
Arm and leg paralysed on right.	Arm and leg in spasm on right.

Third Degree.

But there is a still further degree of hemiplegia: a very grave lesion in the region of the corpus striatum of *one* side will produce palsy of *both* sides of the body; then, of course, the term 'hemiplegia' is, strictly speaking, a misnomer. Similarly, the convulsion which begins on one side—in the hand, for example—will spread at length to the other side; it *becomes* universal. I believe, however, that a fit which thus *becomes universal* depends on discharge in but *one* hemisphere; for facts seem to me to show that each half of the brain represents movements of both sides of the body,[1] but that it represents the movements of the two sides in different degrees and orders. I have, however, had few opportunities of watching convulsions which, beginning in one side, have reached the other. One of my patients, whose fits began in his *right* ulnar fingers, said that once, after the right side of the

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[1] See remarks on this point, 'Lancet,' February 15, 1873.

body had been involved, ' it ' went across his chest to the *left*
arm ; it went *down that* arm, but did not reach the fingers.[1]
One dare not trust much to a patient's observation on such
a matter ; but, as a hint, it is of very great value.

The points I wish to observe about the *second* side are—

1. Is the arm or the leg first affected ?

2. What part of either of the two limbs does the spasm
first reach ? Does it first reach their upper parts (shoulder
and thigh), or their lower parts (hand and foot) ?

3. Does it especially affect any group of muscles—e.g. the
extensors or flexors ?

There are all degrees of palsy, from destroying lesions in
one-half of the brain and there are corresponding degrees of
convulsion ; the above examples are taken simply for illustra-
tion. Another thing we have neglected is the *order of spreading*
of the spasm. Still another is that the bilateral muscles of
both sides are involved when the unilateral of but one side
are well engaged. The following extracts from the report
of a case will show my meaning. There was injury to the
head ; the *first* set of motor symptoms was, palsy of the
right arm and turning of both eyes and of the head to *the
left* ; a second set came on later.

The case is of interest for several purposes of this paper.
It was published [2] under the title, ' *Case of Corpus Striatum
Epilepsy (Hemi-spasm)*,' and chiefly, as will be seen, to illus-
trate Broadbent's Hypothesis.[3]

'There was blood in the arachnoid "cavity" on the left side.
The bulk of it, however, lay in one spot over the frontal con-
volutions, and was so placed as, I imagined, to squeeze the
corpus striatum, which body at the autopsy seemed to be
otherwise undamaged.

.   .   .   .

' The right arm was in spasm, so was the right cheek ; so
was the right orbicularis palpebrarum, and the head and eyes
were turned to the right. So far, then, omitting considera-
tion of the leg, which did not move, the unilateral muscles

[1] See report of his case, ' Med. Times and Gazette,' December 23, 1871.

[2] ' Med. Times and Gazette,' August 15, 1868.

[3] See Broadbent's Paper ' Med. Chir. Rev.,' April 1866. The hypothesis is
stated clearly in Watson's ' Practice of Physic.' See Appendix I. p. 341.

which the left corpus striatum governed were involved. Now for the bilateral muscles which *each* corpus striatum governs in health, which *one* will govern when its fellow is damaged, and which are put in action when *either*, being " unstable," is discharged.

' The bilateral muscles of the thorax were fixed, both masseters were tightened, both cheeks moved (the right being drawn up), and both orbiculares palpebrarum were involved and both sides of the occipito-frontalis acted. With regard to the last-named muscle, its action was, on the right side, partly counteracted by the orbicularis palpebrarum, the eye being closed ; but on the left there was a more even struggle between the two. According to Broadbent's hypothesis, the orbicularis palpebrarum should be most represented in its corresponding (opposite) corpus striatum, as it is a muscle which we can use on one side only ; but still it will be represented in each, as we generally close both eyes at the same time. Now I have noted that in some cases of hemiplegia the patient may be able to close the eye on the non-paralysed side alone, but not the eye on the paralysed side, on which side he can only close the eye by a " bilateral effort "— i.e. when closing both. This small fact I had noticed [1] before I had heard Dr. Broadbent's hypothesis, which seems to account so well for the singularity. The differing degree of spasm in the case above given is still further confirmation.'

[1] ' Lond. Hosp. Reports,' 1865, vol. ii. p. 309.

APPENDIXES

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No. I.

THERE are many things in the two foregoing papers which require more development than has been given to them. Most of them would come under an explanation of the superficial paradox, that whereas *destruction of part of a nervous organ may produce no loss of movements* (no obvious or striking loss is of course meant), *discharge of that part may bring about excessive movements*. I have already in many places urged that, without observing the effects of discharging, as well as of destroying, lesions, we cannot methodically investigate the functions of certain parts of the brain. It will be well here to give particular illustrations, and the illustrations of most point will be those I have already used.

Speaking in the Gulstonian Lectures (1869) of Broadbent's Hypothesis (already referred to at p. 338), I remark[1] :—

' This complementary study of the effect of absence of function and " over-function " of nervous organs is important for several reasons, and notably for this reason, that—in one instance at least—the muscles put in action when a nervous organ is " discharged " are not only those palsied when that organ is destroyed.'

I think the principle is of very great importance for direct *clinical* purposes. I give an illustration, and purposely choose one which shows how the application of the principle in question widens our schemes of investigation. There are cases of disease of the ear (attended by discharge) with which epileptic or epileptiform convulsions occur. I believe the accepted explanation would be that these seizures are reflex —the results of irritation starting from the ear. My speculation is that

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[1] ' Lancet ' Abstract, Feb. 27, 1869. Broadbent's principle of the double representation of those movements which are bilateral in the sense that they must act together, or mostly do act together, is a very important contribution to physiology and scientific medicine. I believe, however (as stated p. 334, and for reasons given more at length ' Lancet,' Feb. 15, 1873), that all orders of *movements* of both sides of the body are represented in each side of the brain, the most voluntary very unequally, the more automatic very equally.

the local instability on which they depend is the result of infarction of veins; it is, I suppose a result of a minor degree of the same process as that which leads to cerebral or cerebellar abscess. Now, cerebral abscess from ear disease occurs mostly in the middle lobe of the brain, and I infer that the changes of instability—the discharging lesion—producing convulsions in those who have epilepsies in association with organic disease of the ears are in that lobe also. But in the case of abscess (nerve tissue destroyed) there usually *are no motor symptoms*—no paralysis, or no local palsy at least. We have not, then, a basis for the kind of comparison which was illustrated on p. 336–7. And it might even be assumed that the middle lobe does not contain processes for movements, because there is no loss of movements (no obvious loss is of course meant), from the destroying effects of abscess occurring therein; and as a necessary inference that minute changes in this part could not lead to *development* of movements (convulsion); investigation would be stopped.

But to quote from the report of some remarks [1] I made on the group of cases referred to :—

'It does not follow that, because *destruction* of a part of the nervous system leads to no symptoms, the part destroyed had no functions. The middle lobe of the cerebrum and the cerebellum of course have functions, and there must be symptoms when these parts *discharge*— where the nervous tissue is not destroyed, but is unstable. A man can *do without* a certain quantity of his cerebrum, cerebellum, or corpus striatum, just as he can do without a certain quantity of his lung or his liver. It is altogether a different thing when, instead of being destroyed, a quantity of his nervous system is unstable and discharges occasionally. Hence it comes to be a matter of importance to study as a first step, with all the precision the subject admits of, the seizures which occur with discharge from the ear, especially when the offensiveness of the discharge implies diseased bone. When we have put the symptoms in groups—in some kind of order—we may cautiously speculate further, and see if there be plausibility in fixing the blame for their occurrence on instability of those regions of the brain in which disease of the ear sometimes leads to abscess.'

No. II.

On Evolution of Nervous Centres.

AFTER the above preliminary remarks, which, I trust, show satisfactorily the importance, both for anatomy and physiology as well as for

[1] 'Brit. Med. Journ.' June 26, 1869, p. 591.

clinical medicine, of the *complementary* study of the effects of the *two* kinds of functional lesions, we can consider certain other points alluded to in the foregoing papers. In the first paper (p. 181), there is a quotation to the effect that removal of the corpora striata does not, in the rabbit, cause loss of movements of the limbs. Let us, however, take instead the following much wider illustration, given by Vulpian in his ' Physiology of the Nervous System.' [1] The case is that of a rat from which the cerebral hemispheres, corpora striata, and a great part of the optic thalami had been removed :—

' Vous voyez qu'il est dans l'immobilité la plus complète. Je vais répéter l'expérience que j'ai déjà faite devant vous. Je fais un bruit d'appel avec les lèvres : vous avez vu le rat tressaillir et sursauter brusquement. Les sensations auditives se produisent donc bien encore chez cet animal. Je pince l'extrémité d'une de ses pattes ; il crie aussitôt. Ces cris paraissent bien être des indices de douleur.'

The following is even more important :—

' Voici un jeune lapin auquel on a enlevé aussi les hémisphères cérébraux et les corps striés. Vous le voyez se tenant très-bien dans l'attitude normale, criant d'une façon plaintive lorsqu'on pince un de ses membres ou sa queue, faisant alors quelques pas, puis redevenant immobile. Ce lapin, ainsi que vous pouvez le constater, fait parfois quelques pas d'une façon tout-à-fait spontanée en apparence, et évidemment très-régulière ; le voilà même qui court ; il va se jeter sur le rebord saillant de cette table, et, après un effort pour avancer, il reste de nouveau tout-à-fait immobile.' [2]

I have a double purpose in considering this kind of experiment. (1) To put forward once more a speculation to account for the fact that a destroying lesion of a part may produce no obvious symptoms when a discharging lesion of that part may produce very striking symptoms. (2) To consider an objection to the view that the cerebral hemispheres are the seats of epileptic discharges, which is often inferred from the statement (the correctness of which I will admit) that epileptic fits are producible in animals whose cerebral hemispheres have been removed.

Since a rat or a rabbit, mutilated as described by Vulpian, does make adapted movements when noises are made, or cries when pinched, and since a pigeon, similarly mutilated, turns its head after a moving candle, it is plain enough that in the *lower* centres of these animals there are sensori-motor processes for the adjustment of *very general* movements to *very general* impressions. But the inference is not that these very same kinds of impressions and movements are not also represented in the *higher* parts removed. Rather the very same processes are in

[1] Page 666.
[2] *Op. cit.* p. 680.

the higher centres *re-represented* in greater complexity and speciality. Let us take a still more striking case. A headless frog will rub vinegar off its back with a hind-leg. But can we suppose that these very movements are not also represented in a more special manner in the frog's encephalon ? Co-ordination in the higher centres is the co-ordination in the lower 'carried a stage further.' [1]

The conclusion I have arrived at from the study of cases of disease is, that the higher centres are evolved *out of* the lower—receiving intercalations as they ascend from the spinal cord to the cerebrum. The higher centre re-represents more specially the impressions and movements already represented generally in the one below it. The co-ordinations are continually being re-coordinated ; for example, those of the pons and medulla are re-coordinated in the cerebrum.[2] There are in the lower centres sensori-motor processes for very *general* purposes, but in *their* higher representatives for the more special. A rude symbolisation would be to suppose the pons Varolii to represent the simpler sensori-motor processes of the cord raised to the ' fifth power,' and the cerebral hemispheres these processes suddenly raised again, let us say, to the ' fiftieth power.'

If such be the plan of structure of nervous organs, we can understand

[1] The two following quotations bear on this matter very directly, each from a very different point, however :—

' It does not follow, as it at first seems to do, that feelings are never located in the inferior nervous centres. On the contrary, it may well be that in *lower* types the *homologues of these inferior centres are the* seats of consciousness. The true implication is, that in *any case* the seat of consciousness is that nervous centre to which the *most heterogeneous impressions* are brought ; and it is not improbable that, in the course of nerve evolution, centres that were once the highest are *supplanted* by others in which *co-ordination is carried a stage further*, and which thereupon become the places of feeling, while the centres before predominant become automatic.' (Spencer, ' Psychology,' vol. i. ch. vi. p. 105. No italics in original.)

' Je viens de vous dire que l'influence du cerveau proprement dit sur les mouvements volontaires était d'autant plus grande en apparence que les animaux opérés *appartenaient à une classe plus élevée*. Vous pouvez en juger par vous-mêmes. Voici un chien sur lequel on a détruit en partie un hémisphère cérébral ; il y a une paralysie très-incomplète des membres du côté opposé, et l'animal est très-affaibli. Voici, au contraire, un pigeon sur lequel un hémisphère est entièrement enlevé ; il semble être presque dans son état normal. *L'influence de l'opération serait de moins en moins appréciable, au fur et à mesure qu'on passerait des oiseaux aux reptiles, des reptiles aux batraciens, et de ceux-ci aux poissons.*' (Vulpian, *Op. cit.* p. 677. The parts I have italicised are very important for the whole of this subject.)

[2] ' This progress (see ' Principles of Psychology,' vol. i. p. 67) from co-ordinations that are small and simple to those that are larger and compound, and to those that are still larger and doubly compound, is one of the best instances of that progressive integration of motions, simultaneously becoming more heterogeneous and more definite, which characterises evolution under all its forms.'—HERBERT SPENCER.

how it is that part of a highly evolved organ—part of the brain, for instance—may be wanting, with only a very special and not an obvious loss of movement or faculty. Or returning to our very rough illustration, many terms may be lacking in the highest ranges of evolution without producing loss of any one power, as of x, or of y, or of z, as each of these will be represented in innumerable other remaining terms. There will be loss only of certain highly special or, metaphorically speaking, *delicate*, processes in which x, or y, or z are *leading*. And such a loss could not be very obvious. In this way we can understand how it is that recovery occurs from hemiplegia, notwithstanding that part of the corpus striatum (see p. 319 and 322) is permanently lacking ; the rest of the corpus striatum also represents the very same muscles, although made up into somewhat different movements. We can also understand how it is that a part of the brain near the corpus striatum may be wanting, without obvious loss of movement.

The above stated conclusions seem to me to be essentially the same as those deductively arrived at by Spencer. I give quotations from his work, italicising some parts of them to make prominent their bearing on my subject.

In his ' Psychology,' [1] speaking of the cerebrum and cerebellum, he says : ' We may regard them as organs of *doubly compound co-ordination* —organs which have for their common function the *re-combining* into *larger* groups, and into countless *different* orders, the *already* complex impressions received by the *medulla oblongata* ; and which have the further function of so arranging the *already* complex motor impulses issuing from the *medulla oblongata* as to form those *far more* involved aggregate actions,' &c. . . .

He then suggests that the cerebrum is for ' doubly compound ' co-ordination in Time, whilst the cerebellum is for ' doubly compound ' co-ordination in Space. After stating the evidence, he says : ' There is complete harmony between the hypothesis and the seemingly-strange facts that these centres may be *partially destroyed* without causing *obvious* incapacity, and that they may be *wholly removed* without destroying the ability to co-ordinate *the less complex impressions and acts*.'

But if it be granted that the hypothesis of Evolution accounts for *absence* of loss of movements [2] (of local palsy), in cases of limited destroying lesion of a nervous centre, there comes the question, ' How does the hypothesis account for the *development* of a large mass of movement (spasm) in corresponding limited discharging lesions ? ' A little consideration will show that the plan of structure, which permits *destruction* of part of a highly ' evolved ' nervous organ with-

[1] Vol. i. p. 60.

[2] Absence of obvious or striking loss of movements is of course meant ; for, as already stated, there will be loss of very special or ' delicate movements.'

out any but the most special loss of the movements or ' faculties ' that
organ represents or shares in representing, is the very one which ne-
cessitates the presentation of a great volume of movement when that
part *discharges*. For the more elaborate the structure of a nervous
organ, that is to say, the greater the number of *different* co-ordinations
it effects—the more grey matter there must be, that is to say, the greater
quantity of explosive material. To quote Spencer again :—

'Each vesicle, or each portion of grey matter that establishes a con-
tinuity between the central termini of fibres, is not *merely* a *connecting*
link ; it is *also* a *reservoir* of molecular motion, which it gives out
when disturbed. Hence, if the composition of nerve is determined as
above indicated, it follows that in *proportion to the number, extensiveness,
and complexity of the relations*, simultaneous and successive, that are
formed among different parts of the organism, will be the *quantity* of
molecular motion which the nerve-centres are capable of disengaging.[1]

'. . . . the *quantity* of molecular motion evolved in the nervous
centres will become great in proportion as the nervous relations increase
in *integration and heterogeneity*.' [2]

Hence, in the convolutions there is not only elaborate structure, but,
as a consequence of this, there is a large quantity[3] of explosive material.

Now we can consider the objection that in epilepsy ' les effets
convulsifs de l'excitation bulbaire sont indépendants de l'influence
cérébrale, *ils peuvent être produits avec des caractères identiques quand
les hémisphères du cerveau sont enlevés*.' [4] Kusmaul and Tenner [5]
write of their well-known experiments : ' Convulsions in epileptic at-
tacks sequential to the abstraction of red blood, do not proceed from
the cerebrum properly so-called, but from the motor centres situated
behind the thalami optici, the excitation being induced by a sudden
arrest of nutrition.' Their experiments show that removal of all parts
in rabbits up to the thalami optici does not exercise any influence
upon the production of general convulsions. But I think the specu-
lation I have advanced shows that in less differentiated animals than
man discharge of the lower centres would be enough to produce severe
universal convulsion. For the condition of the mutilated rat and
rabbit (see quotations, p. 343) shows that they have much grey matter
left in the lower centres, the centres not removed. For, as stated, very
general actions are performed by these mutilated animals. I grant,

[1] ' Psychology,' vol. i. page 35. (No italics in original.)

[2] Spencer, *Op. cit.* vol. i. p. 55. (No italics in original.)

[3] The statement, it is to be observed, is not that the material (in health) is
more explosive, but that there is a great quantity of explosive material. I have,
I now think, erred in saying that the more automatic processes are in health more
unstable than the voluntary or special. (St. Andrews Reports, vol. iii., 1870.)

[4] Jaccoud, *Op. cit.* p. 385.

[5] ' Syd. Soc. Trans.' *Op. cit.* p. 69.

however, that it is an argument against my view that the convulsions in the mutilated rabbits are *as violent* as they are in the non-mutilated. But the lower in the scale of animals the less differentiation there is—the *less* the cerebrum has to do with the movements, and the more the pons, medulla and spinal cord have to do with them. The quotation from Vulpian given on p. 344 (footnote) shows this clearly.

We have now to consider how it can be asserted that discharge of but *one* hemisphere can produce convulsion of *both* sides of the body, as I suppose it does in most of those cases commonly called idiopathic epilepsy. I can only speak of this in mere outline. In the ' Lancet' (February 15, 1873) I have advanced reasons for the belief that movements of the *two* sides of the body are represented in *each* side of the brain. Hence there is convulsion of both sides of the body from discharge of but one hemisphere. We saw this at p. 337, when speaking of convulsions beginning in one hand and becoming universal (*of Corpus Striatum Palsy, and Spasm*, 3rd Degree). But there are cases in which the convulsion is nearly universal to *begin with*, and nearly equal on the two sides. Yet it is significant that the spasm of the two sides is rarely, if ever, absolutely contemporaneous, and rarely, if ever, absolutely equal. The first conspicuous movement in the cases where the fit begins most nearly universally (least *one-sidedly*) is usually turning of the head and eyes to one side ; I suppose the discharge to be of some part of the opposite cerebral hemisphere. I believe these cases differ from those simpler ones just mentioned : first, in that the discharge is more sudden and rapid—(the fit does occur more suddenly and is more quickly over) ; second, in that the sensori-motor processes discharged are more highly evolved ; the evidence for the last statement is that loss of consciousness is the *first* thing, or it is lost *very soon* after a most *general and vague warning*.

So far we have spoken of ordinary gross movements. Now, in epilepsies, especially in those in which loss of consciousness is the first thing, or in which it occurs very early, there are 'vital' symptoms as well as spasm of the limbs and trunk. But it is plain that the cerebrum (which I suppose to be discharged in such seizures) represents the ' vital ' processes of the body. Alterations of pulse, respiration and temperature, constipation, vomiting, ' *tache cerebrale*,' occur in some cases of cerebral tumour, and in cases of large cerebral hæmorrhage. These symptoms are owing to affection of the most general or most automatic processes of the body. They shew that the units of the brain do represent (potentially contain) *all the lower processes* of the nervous system, as well as the higher or more special. In cases of epilepsy we have during, or at the beginning of the paroxysm pallor of the face as well as convulsions.

The sensori-motor processes which underlie consciousness will be the most complex and special of all nervous processes ; they will be evolved

out of and re-represent all lower series. ' *The seat of consciousness is that nervous centre to which, mediately or immediately, the most hetero-geneous impressions are brought.*' [1] The statement is not of the most *numerous* impressions, but most heterogeneous (see definition of volun-tary or special movements, p. 317). To the seat or seats of conscious-ness impressions of *all orders* are brought, and from it issue motor impulses of all orders. It will perhaps be safer to limit this remark by saying that ' all gradations will exist between wholly unconscious nervous actions and wholly conscious ones ' (Spencer). It is to be observed, however, in disease we feel pain in the most automatic parts —for example, in cases of colic and angina pectoris.

The facts that those very epilepsies in which consciousness˙is *first* lost, or is lost very early, are the cases in which the convulsion is nearly universal, in which the two sides are more nearly equally convulsed ; and that it is in these cases that there is at the very first much pallor of the face, tend to confirm the conclusion that the sensori-motor pro-cesses concerned in consciousness are evolved out of and potentially contain all other (lower) series. It is, indeed, most significant, whatever the explanation may be, that there are slight cases (*petit mal*) in which, with transient loss of consciousness, there is deep pallor of the face (and body ?) and a slight wave of universal movement.

The following quotation from Spencer has important bearings on several things discussed in this appendix. It is given here particularly to oppose the notion that the sensori-motor processes concerned in con-sciousness are fundamentally different from, and as it were tacked upon lower series. Why during the excitation of any set sensori-motor pro-cesses, will, memory, &c., arise is unknown. The nature of the con-nection betwixt physiology and psychology, is, so far as we can now see, an insoluble problem.

' Memory, Reason, and Feeling, *simultaneously* arise as the *automatic* actions become complex, infrequent, and hesitating ; and Will, arising *at the same time*, is necessitated by the same conditions. As the ad-vance from the simple and indissolubly-coherent psychical changes, to the psychical changes that are involved and dissolubly coherent, is in itself the commencement of Memory, Reason, and Feeling ; so, too, is it in itself the commencement of Will. On passing *from compound reflex actions* to those actions so highly compounded as to be imper-fectly reflex—on passing from the organically determined psychical changes which take place with extreme *rapidity*, to the psychical changes which, not being organically determined, take place with *some deliberation, and therefore consciously ; we pass to a kind of men-tal action which is one of Memory, Reason, Feeling, or Will, according to the side of it we look at.*

[1] Spencer, ' Psychology,' vol. i., p. 105.

' Of this we may be certain, even in anticipation of any special synthesis. For since all modes of consciousness can be nothing else than incidents of the correspondence between the organism and its environment; they must be all different sides of, or different phases of, the co-ordinated groups of changes whereby internal relations are adjusted to external relations. Between the reception of certain impressions and the performance of certain appropriate motions, there is some inner connection. If the inner connection is organised, the action is of the reflex order, either simple or compound; and none of the phenomena of consciousness proper exist. If the inner connection is not organised, then the psychical changes which come between the impressions and motions are conscious ones; *the entire action must have all the essential elements of a conscious action—must simultaneously exhibit Memory, Reason, Feeling, and Will; for there can be no conscious adjustment of an inner to an outer relation without all these being involved.*'[1]

[1] Spencer's 'Psychology,' No. 25, p. 496. (No italics in original).

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